The Newsletter of the Australian and New Zealand Society of Paediatric Dentistry

Synopses

DECEMBER 2003, ISSUE 27

Enamel hypoplasia or amelogenesis imperfecta?

Winning 2002 Undergraduate essay
Mohamed Z. Murad
School of Dentistry, Otago University, New Zealand

Abstract

The differential diagnosis of dental enamel defects in human teeth is a big clinical problem. Is it Enamel Hypoplasia or is it Amelogenesis Imperfecta? The diagnosis can be challenging.

There are three main questions to ask when trying to decide if the condition is Amelogenesis Imperfecta. Is anyone else affected? Are all the teeth affected? Is there a chronological distribution? A diagnosis of AI is based on the family history, generalised enamel hypoplasia in primary and permanent dentition, and the absence of systemic disease. Hypoplastic defects usually are isolated defects or in some cases may be a type of AI.

Clinical reports, scanning electron microscopic (SEM) studies and molecular genetic investigations, have all provided evidence which questions any classification based on phenotype. If this is true, then a classification which relies for its effectiveness on phenotype at whatever level must necessarily be unreliable.

Accurate diagnosis and appreciation of the associated clinical problems in each case enable the institution of early preventive measures and management techniques using a multidisciplinary approach.

Enamel is ectodermally derived and is produced by ameloblasts which differentiate from inner enamel epithelial cells of the enamel organ.[1] Dental enamel is formed in two stages: deposition of organic matrix (secretory phase), and mineralisation (maturation phase). Disturbance of either stage may cause abnormalities of tooth structure, which are particularly important when enamel is involved. A disturbance of matrix deposition produces hypoplasia (hypoplastic defect), characterised by enamel that is irregular in thickness or deficient in structure; the defects may range from small pits or grooves in the enamel surface to gross deficiency of tissue. Disturbance during the second stage of development causes hypocalcification; although the enamel is of normal thickness, part of it, at least, is poorly mineralised.[2]

Hypoplasia affecting the teeth is generally seen as a defective formation of enamel or dentine. It occurs in permanent or deciduous teeth.[3,4] According to Tiecke and co-workers[5], it is very difficult to differentiate the aetiological factors which alter the formation of enamel matrix (hypoplasia) from those which alter the subsequent calcification (hypo-calcification) since these agents may be the same. Colby and associates[6] suggest that any one of these conditions may develop by itself but mostly they occur together.

The extent of the alteration and the time of occurrence are very important for the final diagnosis. There are basically three types of alteration[3]:

1. Disturbance affecting tooth germ during a specific age, thus disturbing all enamel formed at that time (hypoplasia).

Continued on page 3...

THIS ISSUE

President's Report

Due to the time constraints of my short incumbency as President I have been visiting as many provincial Branches as possible over the past winter months. My objective in visiting and talking with members at a branch level is to maintain and generate interest in line with the Society's aim of promoting continuing education and professional interchange in paediatric dentistry for all who provide dental care for children.

I have attempted to provide encouragement to attend both the next (14th) Biennial Convention to be held 18-20 March 2004 in Melbourne, and also to attend and assist with ANZSPD's commitment to sponsor and hold the International Association of Paediatric Dentistry meeting in Sydney in October 2005. Generating interest and enthusiasm for ANZSPD continuing education at Branch level, as well as requesting articles for our Colgate Oral Care sponsored Newsletter *Synopses* has also been a goal. It has been a pleasure to again meet up with old colleagues and friends as well as meeting new acquaintances and making new friends amongst colleagues who are involved in dental care delivery and dental health promotion for children.

I spoke to the South Australian Branch at the ADA Rooms on Tuesday 8th July. After providing an update on paediatric dental traumatology, I strongly encouraged Branch participation with Federal ANZSPD, as well as fielding some questions concerning the long awaited appearance of the next issue of "Synopses". I visited the West Australian Branch on the weekend 26-27 July, where a two day meeting was organised by State and Federal Business Manager/Secretary Alistair Devlin, and Branch President Tim Johnston. After talking for most of Saturday afternoon on topics included in a 'Pot Pourri' of Paediatric Dentistry, I thoroughly enjoyed participating with Branch members who presented and discussed case reports on Sunday morning. I have subsequently recommended this format to my own Branch committee in Victoria.

I visited the Queensland Branch on weekend 23-24 August where I gave presentations complementing the Conference theme of Special Care in Paediatric Dentistry as requested by Branch President Laurie Bourke. I can recommend Queensland hospitality. I also visited Hobart and addressed Tasmanian Branch members on weekend 27-28 September as well as providing support to the 2003 RK Hall Visiting Lecturer, Dr Kim Seow. Dr Seow presented some very interesting research on aetiology and risk factors in early childhood caries. Dr Tasha Dodd and her team are to be commended in providing leadership in our smallest Branch.

Organisation of the 14th ANZSPD Biennial Conference to be held in Melbourne 18-20 March 2004 is well underway. The Local Organising Committee consists of the Victorian Branch executive, Mala Desai, Jodie Heap, Karen Kan, Chris Olsen, John Sheahan, Donna Tomeski and Felicity Wardlaw. The Scientific Programme Subcommittee comprises of Mala Desai, Karen Kan, Nicky Kilpatrick, Louise Brearley Messer, Chris Olsen and John Sheahan. The professional conference organiser 'Happenings Australia' has been engaged to assist in holding of the Conference.

The Melbourne Exhibition and Convention Centre, Southgate, has been booked for the conference. The Gala Dinner is to be held on Friday evening, 19 March, at the River Room, Crown Casino, located opposite the conference venue. Keynote Speaker is Dr Stephen Fayle from Leeds Dental Institute, UK, supported by a number of prominent local Australian and New Zealand speakers.

The theme for the Conference is "Tomorrow's Teeth for Toddlers, Teens and In betweens" The Scientific Programme complimenting this theme includes topics:

- Behaviour Management: Hug'em, drug'em or slug'em.
- Prophylactic Antibiotic Cover: Who are we really protecting?
- Prevention of Dental Disease: New modalities for a new generation.
- Fluoride: Getting the balance right.
- Erosion: Is my child at risk? Prevention, early diagnosis, and restoration.
- Molar – Incisor Hypoplasia: Management.
- The First Permanent Molar. An orthodontist's perspective.

Colgate Oral Care, in continuing a long association in supporting ANZSPD activities has agreed to be Principal Sponsor and as in the past also sponsor the Colgate Postgraduate Research Award, a well as sponsor the Welcome Reception. Again by the time that members read this editorial Expression of Interest and Conference Registration brochures will have been circulated to members and other groups with an interest in paediatric dentistry.

I must make mention that the Australasian Academy of Paediatric Dentistry at its Meeting in Sydney on 13th September launched the copyrighted 'Standards of Care in Paediatric Dentistry'. A number of ANZSPD members contributed to this important document, which provides guidelines of care, which will be especially useful should controversy arise over treatment issues in Australia, New Zealand and elsewhere.

I take this opportunity to acknowledge Dr Karen Kan, who has taken over the key role as Editor of Synopses. Karen brings to the position both much needed enthusiasm and the necessary IT skills to facilitate further development of the Newsletter. Councillors are requested to provide State reports promptly, and also to encourage their members to submit articles. Issue 26 June 2003 was mailed out in early August. It is understandably difficult for the Editor to produce new editions when material is not provided on time. In addition, thanks are due to Dr John Winters who provided a draft of the proposed ANZSPD Website at the ANZSPD Federal Council Meeting held in Sydney

on 14th September. It is planned to have the website and links up and running in the near future.

I make special mention of our hardworking and long serving Business Manager/Secretary Alistair Devlin, without whose endeavours the Society would not function. It must be acknowledged that while Presidents come and go Alistair remains in this crucial but often thankless role of providing the continuity to keep ANZSPD on course.

I am looking forward to meeting with you all at the 14th ANZSPD Biennial Conference on 18-20 March next year.

Chris Olsen

Continued from page 1...

2. Hereditary disturbances, which alter all enamel matrix independently (amelogenesis imperfecta).
3. Individual alterations, which occur in one or more teeth.

Thoma and Goldman[7] believe that hypoplasia is due to an alteration in ameloblastic activity of the enamel organ. This alteration is probably inhibitory in nature and causes atrophy and lack of function of ameloblastic cells. These cells do not recover their vitality and when the alteration disappears, neighboring cells continue to form normal enamel, leaving behind a line of ill-formed enamel.

The exact cause of enamel defect is often not obvious from the clinical history, but a number of potential causes have been identified. (Table 1).

Hereditary enamel defects (amelogenesis imperfecta) affect all areas of the enamel in permanent and deciduous teeth, in contrast to the ordinary type of 'chronological' enamel hypoplasia.[8]

The frequency of enamel hypoplasia is greatest in the anterior dentition, is intermediate in premolars, and lowest in molars.[9] On average, maxillary central incisors are most often hypoplastic and canines, lower lateral incisors, and second molars are least often hypoplastic.[10]

Within the dental literature one hypothesis is repeatedly given to explain variations in frequencies of defects by tooth type. According to this 'time of development' hypothesis, the frequency of defects is dependent on which tooth crowns are developing at the time which insults are most active. Earlier-occurring insults will affect only the earlier developing tooth crowns, whereas later-occurring insults will affect subsequently developing tooth crowns.[11] Analysis of tooth crowns by developmental period clearly shows that time of development is not the sole determinant of hypoplasias.[11,12] (Fig. 1)

While time of development provides an explanation for part of the difference in frequency of hypoplasias, it clearly does not explain all of the differences, as teeth developing at the same time do not all have a similar frequency of enamel hypoplasia. Therefore, factors should also be considered which might govern susceptibility to hypoplasias such as biological gradients.[11]

Hypoplasias are not randomly distributed within tooth crowns. Furthermore, the distribution is similar for all tooth crowns, regardless of time of development. Overall, there is a consistent pattern of increased frequency of defects in the middle thirds of tooth crowns. While the cervical third is more hypoplastic than the incisal third in all tooth classes, there is some evidence that this difference increases as one moves toward the posterior teeth.[11] (Fig. 2)

The process, which controls within-tooth differential susceptibility to hypoplasias, is yet to be considered. However study by Goodman and Armelagos (1985) suggested four factors to considered. First, the number of secretory (matrix-forming) ameloblasts may decrease towards the cervical half of the crown. Second, there is some evidence that rate of crown development increases in the middle and cervical third of tooth crowns. In this situation ameloblasts are forming more enamel in a given unit of time and may, therefore, be rendered more vulnerable to disruption when their rate of development is rapid. A third explanation holds that the expression of a hypoplasia is a function of enamel prism direction. Enamel prisms are most nearly perpendicular to the surface at the zone just cervical to the midpoint. This orientation may increase the ease with which one can discern variation in prism lengths. Fourth, longer prism might be more sensitive to disruption.[11]

When long-term ingestion of excessive fluoride is the cause of hypoplasia, the affected enamel can be expected to be distributed over the part of the crowns of teeth developing during that period.[2]

Fig.1 The parts of the crowns of developing permanent teeth that would be expected to be affected by systemic disturbances from birth to seven years.[12]

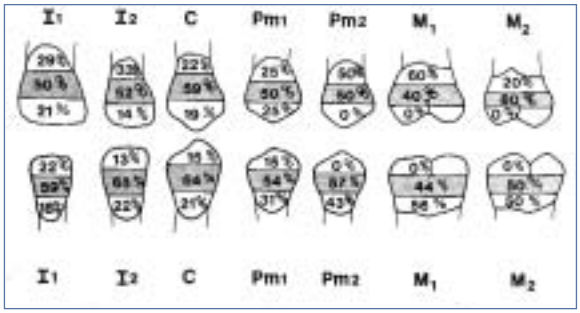

Fig.2 The frequency of enamel hypoplasia by incisal/occlusal, mesial, and cervical crown thirds by tooth.[11]

Table (1) Causes and factors commonly reportecd to be associated with enamel hypoplasias of human teeth

<table>
<tr><th colspan="2">I. Chronological enamel defect</th><th>II. Hereditary enamel defect</th></tr>
<tr><th>Local causes</th><th>Systemic causes</th><th>Genetic causes</th></tr>
<tr><td>Local acute mechanical trauma
■ Neonatal Mechanical Ventilation (Moylan et al., 1980)
■ Falls and Similar Injuries (Andreasen et al. 1971)
■ Gunshot (Pindborg,1970)
■ Surgery (Williamson, 1966)
■ Ritual Mutilation (Pindborg, 1969)</td><td>Inborn errors of metabolism
■ Galactosaemia (Benusis et al., 1978)
■ Phenylketonuria (Meyers et al, 1968)
■ Alkaptonuria (Siekert and Gibilisco, 1970; Link, 1973)
■ Erythropoietic Porphyria (Rayne, 1967)
■ Primary Hyperoxaluria (Glass, 1967)</td><td rowspan="2">Amelogenesis Imperfecta (AI) as isolated phenomenon (Table. 2)</td></tr>
<tr><td>Electric burn (Alexander, 1964)</td><td>Chromosomal anomalies
■ Trisomy 21 (Down syndrome). (Cohen, 1971)</td></tr>
<tr><td>Irradiation (Doline et al., 1980)</td><td>Congenital defects
■ Heart disease (Hakala, 1967)
■ Unilateral Facial Hypoplasia (Gibbard and Winter, 1972)
■ Unilateral Facial Hypertrophy (Benusis et al., 1978)</td><td rowspan="4">Amelogenesis Imperfecta (AI) associated with other lesions
■ Epidermolysis bullosa. (Arwill et al., 1965)
■ Pseudohypoparathyroidism. (Jensen, 1981)
■ Taurodontism, curly hair, sclerotic bones. (Tricho-dento-osseous syndrome). (Gorlin et al., 1976)</td></tr>
<tr><td rowspan="3">Local infection
■ Periapical Osteitis (McCormick and Filostrat, 1967)
■ Acute Neonatal Maxillitis (Poley and Wegner, 1967)</td><td>Neonatal disturbances
■ Premature birth (Rosentein, 1974)
■ Hypocalcaemia (Purvis et al., 1973)
■ Haemolytic anaemia (Solyga, 1970)
■ Allergy (Rattner and Meyers, 1962)</td></tr>
<tr><td>Infection diseases
■ Viral Rubella (Guggenheim et al., 1971)
■ Bacterial Syphilis, Tetanus</td></tr>
<tr><td>Neurological disturbances
■ Tuberous sclerosis (Hoff et al., 1975).</td></tr>
<tr><td rowspan="6">Regional Odontodysplasia
(Gardner and Sapp, 1973)</td><td>Endocrinopathies
■ Hypothyroidism.
■ Hypoparathyroidism (APECS). (Myllarniemi et al., 1978)
■ Diabetes (Grahnen et al., 1968)</td><td rowspan="6">Other genetically determined diseases
■ Ehlers-Danlos Syndrome. (Barabas, 1969)</td></tr>
<tr><td>Nutritional deficiencies.
(Enwonwu, 1973)</td></tr>
<tr><td>Nephropathies
■ Nephrotic syndrome (Oliver et al., 1963)
■ Infections of urinary system (Freden et al., 1980)</td></tr>
<tr><td>Enteropathies
■ Coeliac disease (Smith et al., 1979)
■ Lymphangiectasia (Dummer, 1977)</td></tr>
<tr><td>Liver disease. (Pindborg, 1970)</td></tr>
<tr><td>Intoxications
■ Tetracyclines (Baker, 1975)
■ Thalidomide (Axrup et al., 1966)
■ Vitamin D (Pindborg, 1970)
■ Pica. (Lawson et al., 1971)</td></tr>
</table>

Modified from Pindborg. 1982.[8]

Table (2) Classification of Amelogenesis Imperfecta (AI) according to Witkop (1989). Clinical and radiographic appearance

Inheritance	Type	Subtype	Colour	Enamel Thickness	Enamel hardness	Clinical appearance	Radiographic appearance
Hypoplastic (HAI)	I	Enamel does not develop to normal thickness					
Autosomal-dominant	IA	Pitted	Yellow-white	Normal	Normal	Pin-points in random, multiple teeth	Mild lucency in deep pits
	IB	Local	Yellow-white	Normal	Normal	Pits or depressions, usually buccally, linear horizontal	Mild lucency in deep pits
	ID	Smooth	White to yellow-brown	¼ to 1/8th of normal	Normal but may abrade	Thin, glossy, general; teeth do not contact. Anterior Open Bite (AOB) occurs in a bout 50%.	Thin, opaque enamel; normal contrast to dentine
	IF	Rough	Yellow-white to white	¼ of normal	Chips from dentine	Rough, granular surface; teeth do not contact. (AOB) occurs in a bout 50%.	Thin, opaque enamel; normal contrast to dentine
Autosomal-recessive	IC	Local	More severe than the dominant type.			Hypocalcified enamel may occur in the hypoplastic areas.	
	IG	Rough	Yellow	Nearly absent		Rough, granular surface; occasionally missing teeth. (AOB) occurs frequently 11/13	Enamel not evident
X-linked dominant	IE	Smooth Male	Yellow-brown	Thin	Abrades easily	Smooth, shiny, thin; teeth do not contact. (AOB) most common presentation for affected males.	Thin, opaque enamel outline; normal contrast to dentine
		Smooth Female	Yellow	Normal and thin	Abrades easily	Vertical bands of normal enamel between hypoplastic (AOB) occurs in about 1/3 of the affected females.	Vertical radiolucent areas
Hypomaturation (HMAI)	II	Enamel is of normal thickness but has a mottled appearance.					
Autosomal-recessive	IIA	Pigmented	Brown; stains deep	Normal	Chips easily	Shiny, smooth, dark enamel. (AOB) occurs infrequently.	Enamel same radiodensity as dentine
X-linked -recessive	IIB	Male	White; darkens with age	Near normal	Soft; abrades	Mottled enamel, which darkens; posterior cervical less affected	Enamel same radiodensity as dentine
		Female	Yellow	Normal	Soft; abrades	Vertical bands of normal enamel between abnormal; posterior cervical less affected	Enamel same radiodensity as dentine
Autosomal-dominant	IIC	Snow-capped teeth	Opaque white	Involve ¼ to 1/3 of the crowns of teeth, it maybe mistaken for fluorosis but does not have the accented perichymata linear pattern seen in fluorosis.			
Hypocalcification (HCAI)	III	Enamel initially develops normal thickness, but consists of poorly calcified matrix, which is rapidly lost leaving dentine cores.					
Autosomal-dominant	IIIA	Regular	White to honey	Normal	Soft; cheesy	Soft, cheesy enamel; can be removed with a prophylaxis	Enamel same as or less radiodensity than dentine.
Autosomal-recessive	IIIB	Regular	Same clinical findings as IIIA, but with a greater degree or severity				
Hypomaturation-Hypoplastic with taurodontism.	IV	Enamel is a mottled white-yellow-brown with pits most frequently on the labial face or is thin with areas of Hypomaturation.					
Autosomal-dominant	IVA	HM-HP with taurodontism		Near normal		Enamel predominantly Hypomaturation with areas of Hypoplastic	Enamel same as radiodensity than dentine.
	IVB	HP-HM with taurodontism		Thin		Enamel predominantly Hypoplastic with areas of Hypomaturation	

Modified from Witkop (1988)[13] and Sengun et al. 2002[17].

What is Amelogenesis Imperfecta?

Amelogenesis Imperfecta (AI) is a group of inherited abnormalities of dental enamel.[13,14] It may be differentiated into three main groups: hypoplastic (HP), hypocalcified (HC), and hypomature (HM), depending on the clinical presentation of the defects and the likely stage of enamel formation that is primarily affected.[13] Each main clinical group of AI may be further divided into several subgroups depending on the mode of inheritance, as well as the clinical appearance of the defective enamel,[13,14] although in some cases, overlapping clinical features may make distinction difficult.[15] (Table 2).

The prevalence of this condition has been estimated to range from (1 in 718)[17] to (1 in 14,000)[18], depending on the population studied. Hypoplastic AI represents 60-73 per cent of all cases, hypomaturation AI represents 20-40 per cent, and hypocalcification AI represents 7 per cent.[19]

In studies made of the prevalence of anterior open bite in patients with amelogenesis imperfecta, it was noted that it occurred in 24 per cent in the affected group compared with only 2 per cent in the world population. The coexistence of the two conditions can be due to a pleiotropic action of the AI genes, influencing the growth of the craniofacial skeleton.[20] Disorders of the enamel epithelium also can cause alterations in the eruption mechanism, resulting in the anterior open bite.[21] However, Witkop and Sauk (1976)[22] suggested that this malocclusion was of a dentoalveolar nature, due to the patient inserting his tongue in a reaction to protect against aggressor thermal stimuli, resulting in local interference that would prevent alveolar growth.

Comparing the cephalometric radiographs of AI patients with a control group, other discrepancies in the facial skeleton are found, such as a reduction in the angle of the mandibular plane, increased anterior facial height and a reduction in the posterior portion of the skull base, suggesting that the open bite may be of skeletal origin and not due to a disorder in the dental eruption mechanism.[21] Investigations into the embryonic development of the craniofacial complex suggest that this and the dental enamel have a common origin. In AI, therefore, it is possible that the gene acts in cells derived from the neural ridge, causing subsequent anomalies in the dental enamel and in the skull.[20]

The differential diagnosis of dental enamel defects

The differential diagnosis between enamel hypoplasias and Amelogenesis Imperfecta must be based on clinical and, if possible, laboratory data. This diagnosis can be the key to discovering genetic and systemic diseases, and also local aggressor factors that occur during dental development.[23] The diagnosis of AI should be based on the following criteria: 1) generalised enamel hypoplasia of both the primary and permanent dentitions; 2) family history of the condition, although in the recessive forms, or with new mutations, there may be no previous history; 3) absence of systemic diseases that may cause generalised enamel hypoplasia resembling AI (e.g. systemic disorders involving calcium metabolism such as renal and liver disorders).[23,24]

Although recent research has made significant advances into the diagnosis of different types of AI by molecular[25-27] and biochemical[28] methods, these sophisticated techniques are not yet routinely available.

Molecular genetic studies

Molecular genetic studies have shown that the aetiology of AI is related to the alteration of genes involved in the process of formation and maturation of the enamel.[29] Although the genetic origin of the autosomal forms is less understood, analysis of X-linked AI has shown the defective gene for this specific AI type to be closely linked to the locus DXS85 at Xp22.[30] Interestingly, this also has been identified as the general location of the human gene for amelogenin, the principle protein in developing enamel.[31,32] Information from molecular genetic studies will ultimately lead to identification of the genes involved in normal and pathological enamel formation and provide a basis for definitive diagnostic tests.

Biochemical analysis of enamel protein

Normal mature human enamel is reported to contain between 0.01 per cent to 1.0 per cent protein by weight with some areas such as the cervical enamel and that closest to the dentinoenamel junction having greater than 0.6 per cent protein.[33-36] Analysis of autosomal recessive hypomaturation AI showed a protein content of approximately five per cent by weight in the fully developed enamel, while hypoplastic AI teeth contained two per cent protein.[28] The amino acid profiles of both normal and AI enamel were similar although there appeared to be increased amounts of glycine in the AI enamel. Hypoplastic AI enamel showed an amino acid profile similar to normal primary enamel in contrast to hypomaturation AI that exhibits an amelogenin-like character.[37] The protein content of fully developed enamel from different AI types appears quantitatively and qualitatively different (Table 3)[37]. This should allow discrimination of AI types based on enamel composition along with the clinical, hereditary, and histological features. Furthermore, the distinct differences in amino acid profiles of the enamel protein seen in different AI types provides an objective biological marker that appears useful in delineating the different AI types.[28,37,38]

Currently, diagnosis of the different AI variants rests mainly on the clinical presentations and their modes of inheritance as determined from family pedigrees. Accurate diagnosis is clinically important for several reasons. First, it is important to exclude the presence of certain systemic diseases that may show generalised enamel hypoplasia as an accompanying sign.[23,24,39] Second, accurate diagnosis enables genetic counselling,[40] which is often sought by affected families. Third, accurate diagnosis leads to the recognition of clinical problems that are associated with the condition, and so preventive measures may be instituted early. Fourth, diagnostic differentiation of the many variants of AI may help to determine the type of restorations[41,42] that are most successful.

Diagnostic difficulties

Witkop's classification of AI types (Table 2) may cause difficulties in identifying some variants that simultaneously show clinical features of two or more groups (e.g., hypoplasia is often noted in the hypocalcified groups).[29] Overlapping features also have been identified both micromorphologically[15,43] and microradiologically.[44,45] In addition, the unavailability of dental data from

certain family members, as well as incompleteness of pedigree, may compromise accurate diagnosis in many AI patients.[29]

Management strategies for enamel hypoplasia

There are many manifestations of enamel hypoplasia and there is no standard formula for successful treatment.[46] Hypoplastic defects vary greatly in their size and shape, but teeth commonly have a less than optimal crown form. Adequate restoration of any deficit of enamel is often difficult, as the deficit may not follow the 'classical-caries' pattern.[47] In treating hypoplasia the degree of alteration preparation varies from mild, as in home bleaching, to severe – e.g. the provision of ceramometal crowns. With arrival of adhesive technology the range of treatment possibilities has expanded.[47]

Treatment options

The aims of treatment of affected hypoplastic teeth are[47]:

- To alleviate any pain, sensitivity or infection
- To preserve pulp health
- To maintain the occlusion
- To improve the appearance of the defects

Hypoplastic teeth may be treated conservatively[48-50] by:

- Dental bleaching;
- Conservative enamel reduction (macroabrasion); or
- Enamel microabradsion via an acid abrasive paste.

One of the most commonly and severely affected teeth is the first permanent molar. Fortunately, modern dental materials provide a number of ways of restoring compromised first permanent molar teeth but, in some instances, the best treatment of hypoplastic teeth are their timely extraction[47]. In general, the treatment of isolated hypoplastic defects depends on the severity of the defect, and the degree of cooperation and the age of the child.[47] (Table 4)

Restoration and materials

Management of enamel hypoplasia depends on the patient's treatment needs and the defects of the hard tissues. Biological, biomechanical, economical and psychological considerations must also be taken into consideration.[47] (Table 5)

Restoration with glass ionomer cement (GIC)

The simplest way to restore a minimally to moderately affected hypoplastic teeth is to use GIC; the greatest advantages for the use of GIC are its release of fluoride and chemical bond of the material to the enamel and dentine, thus allowing minimal cavity preparation.[51] Some report that the long-term survival of GIC restorations in non-hypoplastic teeth is inferior to other materials[52]; therefore GIC should be used as an interim restoration until the decision to place a more permanent restoration has been made.

Restoration with composite resin (direct composite)

Good aesthetic and long-term results utilising composite resin restorations in the treatment of hypoplastic defects have been reported.[53,54] The micro-mechanical bond of composite resin to enamel achieved by acid etching minimises the amount of tooth preparation required to restore hypoplastic teeth. In addition, sodium hypochlorite (NaOCl) is known to be an excellent protein denaturant which should be capable of removing excess enamel protein and enhances the bonding.[47,55] Newer restorative techniques may aid in the restoration of hypoplastic teeth. Air-abrasion technology has become more widely used in the last decade. It is an effective pre-treatment for sealant placement and in concert with phosphoric acid treatment significantly enhanced the long-term bond of a sealant to enamel.[56]

Partial-and full-coverage restorations

Indirect adhesive or cast restorations (non-precious alloy, gold, and composite)

For moderate enamel hypoplasia, an indirect restoration may be contemplated in the late mixed and permanent dentition for an affected first permanent molar. This type of restoration not only protects the underlying tooth structure, but also maintains function, does not encroach on the periodontium, and adequately controls any sensitivity that is commonly present with hypoplastic teeth.[47]

Stainless steel crowns

Full-coverage crowns are the treatment of choice for moderate to severely hypoplastic teeth.[56] The objectives of a stainless-steel crown placed on a hypoplastic tooth are to create a proper occlusal relationship, establish correct interproximal contacts, and control symptoms such as tooth sensitivity.[56,57] When a preformed stainless-steel crown is adapted and cemented properly on a carefully prepared tooth, it is durable, reliable and functional for many years, usually requiring no additional intervention other than periodic radiographs and review.

Extraction of affected teeth

If single or multiple first permanent molars with extensive hypoplasia are present, consideration of timely extraction of the affected tooth or teeth may be advantageous even if root-canal treatment can be performed.[58] The suggested best time to extract a first permanent molar is between age 8.5 and 10.5 years, which should coincide with the calcification of bifurcation of the second molar. Other considerations include facial profile, degree of crowding, congenital absence of permanent teeth, and other orthodontic considerations.[59,60]

Laser therapy

Lasers are now more widely used for oral hard and soft-tissue treatment. Hashimoto[61] reported that after irradiation of defective enamel of rats with an Nd:YAG laser, the surface became smooth and small defects disappeared.

The Multidisciplinary Approach in managing Amelogenesis Imperfecta

Management of AI is more complex than enamel hypoplasias, as all teeth are almost affected equally and different complications that may be encountered in each type of AI. So comprehensive oral rehabilitation is required taking in

Table (3) Amino acid analyses of normal and AI enamel

Amino Acid	Normal Primary	Normal Permanent	Hypoplastic AI	Hypomaturation AI
Cystic acid	0	0	0	4.2
OH-proline	0	0	0	0
Aspartic acid	83.2	93.5	64.5	38.6
Threonine	41.0	48.4	28.7	36.2
Serine	83.4	127.2	54.1	79.2
Glutamic acid	152.0	124.1	119.8	122.8
Proline	84.3	138.8	98.1	196.8
Glycine	211.7	159.7	298.8	95.4
Alanine	76.5	61.9	106.5	21.1
Cystine	0	0	0	0
Valine	30.4	44.8	30.8	33.1
Methionine	0	0	0	28.3
Isoleucine	21.3	22.4	12.5	29.5
Leucine	58.4	62.2	39.4	80.3
Tyrosine	26.5	8.6	17.5	83.2
Phenylalanine	27.3	28.3	21.7	32.6
OH-lysine	0	0	0	0
Lysine	27.1	44.0	33.4	29.7
Histidine	42.6	20.8	25.6	51.8
Arginine	33.0	24.3	46.7	36.4

account the restorative and orthodontic consideration.[62] Numerous treatments have been described for rehabilitation of amelogenesis imperfecta in adults[63-65] and children.[66-69]

The severity of dental problems varies with each type of AI. The hypoplastic variants tend to be associated with less severe clinical problems, with the mildest problems encountered in the pitted hypoplastic type of AI. By contrast the patients with the hypocalcified type of AI usually presented with the most severe clinical problems.[29]

Clinical problems associated with Amelogenesis Imperfecta

- Poor dental aesthetics

Poor dental aesthetics are usually the result of surface roughness, staining, and abnormal crown shapes from enamel loss. Several strategies may be used to overcome the compromised aesthetics. In patients with hypoplastic type of AI, there is usually sufficient enamel available for bonding so that composite resins veneers may be used to mask the staining and improve the crown morphology. However, in patients affected by the hypocalcified varieties of AI, enamel is usually insufficient for direct bonding, and dentine bonding resins[70] or glass ionomer cements[71] are first required to bond to the underlying dentine before applying the veneer of composite resins. Other anterior veneers using porcelain are also likely to be useful, particularly if sufficient enamel is available for bonding.[29]

Porcelain jacket crowns, which provide aesthetic permanent restorations, are probably the restoration of choice for affected adults with AI, but their use in young patients is contraindicated due to presence of large pulps.[29] In the primary dentition, anterior primary teeth may be restored with strip crowns; alternatively, anterior stainless steel crowns with composite resin facings have been tried successfully.[72]

- Dental sensitivity
- Dental caries
- Gingival inflammation
- Anterior open bite

Alteration of the occlusal vertical dimensions may occur in AI. Anterior open bite has been associated in AI, particularly in the hypocalcified types.[14, 68,73,74]

Types of corrective treatment that have been suggested range from routine orthodontic banding[75] to orthognathic surgery[76], all with varying degree of success.

In contrast to anterior open bite, collapse of the posterior occlusal segments, leading to deep anterior overbite also has been reported in some types of AI.[14] Loss of occlusal vertical dimension is best prevented as early as possible, preferably in the primary dentition by placing posterior steel crowns.[77] In the case of patients who have lost extensive interocclusal height, rehabilitation may be achieved by posterior full crowns and/or by overlay dentures.[78,79]

Other clinical problems that have been reported include delayed eruption and/or tooth impaction[70], resorption of unerupted teeth[80], and follicular cysts associated with impacted teeth.[15]

Table (4) Summary of treatment for hypoplastic teeth[2]

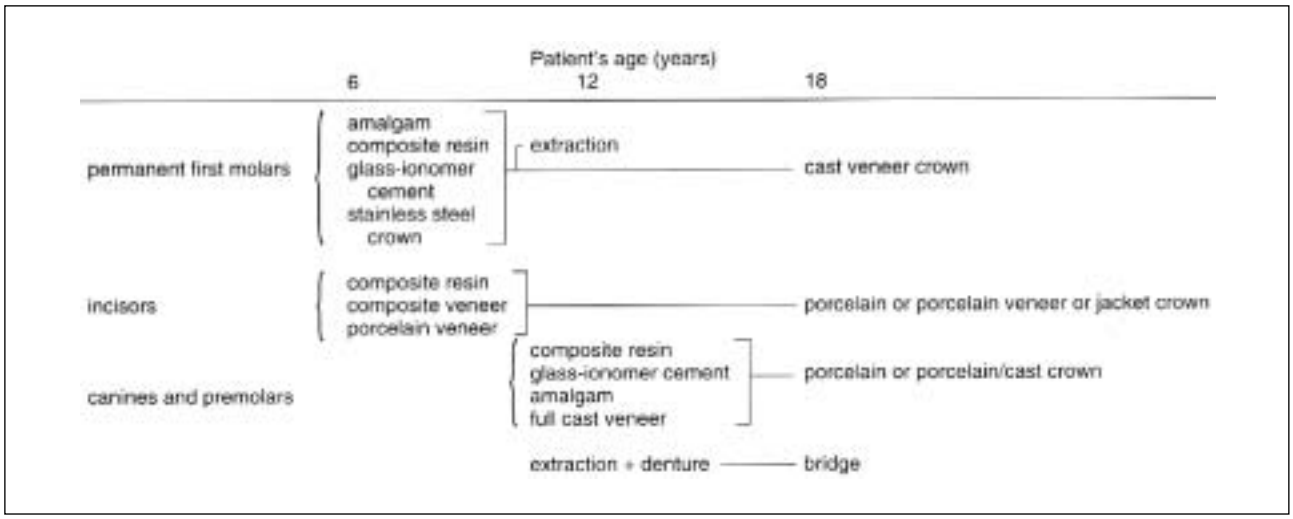

Table (5) Summary of materials and treatment of enamel hypoplasia[47]

Degree of hypoplasia	Restorative material and treatment	Further treatment
Minimal: **< 1/3 of occlusal surface**	Temporary measure to gain cooperation GIC to fill defect	3-monthly monitoring until co-operation improves
	Permanent restoration: GIC and fissure sealant Composite resin and lining	 6-monthly follow-up 6-monthly follow-up
Moderately affected: **1/3-2/3 of occlusal surface**	Temporary measure to gain cooperation GIC to fill defect	 As above
	Permanent restoration: Composite resin and GIC dentine lining Cast restoration or indirect composite resin restoration Stainless-steel crown	 As above 6-monthly to yearly follow-up and radiographs yearly 6-monthly to yearly follow-up and radiographs yearly may contemplate extraction of tooth as part of orthodontic treatment. Replacement in late adolescence
Severely affected **>2/3 of occlusal surface**	Stainless-steel crown Extraction	As above Yearly follow-up to monitor eruption of permanent teeth

Conclusion

The rehabilitation of amelogenesis imperfecta must take into account the development of the child's teeth, the health of periodontal tissues, and the mandibular and maxillary growth. Accurate diagnosis and appreciation of associated clinical problems in each case enable the institution of early preventive measures and management techniques using a multidisciplinary approach.

References

1. McDonald RE, Avery DR. *Dentistry for the child and adolescent*. St Louis: Mosby, seventh edition, 2000: 115-122.
2. Andlaw RJ, Rock WP. *A manual of paediatric dentistry*. Churchill Livingstone, fourth edition, 1996: 141-148.
3. Alvares LC. de Souza Freitas JA. *Hypoplasia and hypocalcification of enamel. Report of a case*. Oral Surgery, Oral Medicine, Oral Pathology. 1969; 28 (1): 73-5.
4. Dummer PMH, Kingdon A, Kingdon R. *Distribution of developmental defects of tooth enamel by tooth-type in 11-12-year-old children in South Wales*. Community Dentistry and Oral Epidemiology 1986; 14: 341-344.
5. Tiecke RW, Stuteville OH, Calandra JC. *Pathology Physiology of Oral Disease*, St Louis, 1959, The C.V. Mosby Company.
6. Colby RA, Kerr DA, Robinson HBG. *Color Atlas of Oral Pathology*, Philadelphia, 1956, J.B. Lippincott Company.
7. Thoma KH, Goldman HM. *Oral Pathology*, fifth edition, St. Louis, 1960, The C.V. Mosby Company.
8. Pindborg JJ. *Aetiology of developmental enamel defects not related to fluorosis*. International Dental Journal. 1982; 32(2): 123-34.
9. El-Najjar MY, DeSanti MV, Ozebek L. *Prevalence and possible etiology of dental enamel hypoplasia*. American Journal of Physical Anthropology. 1978; 48(2): 185-92.
10. Cutress TW, Suckling, GW. *The assessment of non-carious defects of enamel*. International Dental Journal.1982; 32:117-122.

11. Goodman AH, Armelagos GJ. *Factors affecting the distribution of enamel hypoplasia within the human permanent dentition*. American Journal of Physical Anthropology. 1985; 68 (4): 479-93.
12. Hall RK. *Pediatric orofacial medicine and pathology*. Chapman & Hall, London, ch 11. 1994.
13. Witkop CJJr. *Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: problems in classification*. Journal of Oral Pathology. 1988; 17(9-10): 547-53.
14. Sundell S, Koch G. *Hereditary amelogenesis imperfecta. I. Epidemiology and clinical classification in a Swedish child population*. Swedish Dental Journal. 1985; 9(4): 157-69.
15. Seow WK. *Dental development in amelogenesis imperfecta: a controlled study*. Pediatric Dentistry. 1995; 17(1): 26-30.
16. Sengun A. Ozer F. *Restoring function and esthetics in a patient with amelogenesis imperfecta: a case report*. Quintessence International. 2002; 33(3): 199-204.
17. Bäckman B, Holm AK. *Amelogenesis imperfecta: prevalence and incidence in a northern Swedish country*. Community Dental Oral Epidemiology. 1986; 14:43-47.
18. Witkop CJJr, Sauk JJJr: *Heritable defects of enamel*. In Oral Facial Genetics, Stewart RE, Prescott GH (eds), and St Louis: CV Mosby Co., 1976: 151-226.
19. Pearce K. *Amelogenesis imperfecta: from pedigree to practice*. Synopses (ANZSPD). 2001; 25:11-14.
20. Rowley R. Hill FJ. Winter GB. *An investigation of the association between anterior open-bite and amelogenesis imperfecta*. American Journal of Orthodontics. 1982; 81(3): 229-35
21. Persson M, Sundell S. *Facial morphology and open bite deformity in amelogenesis imperfecta. A roentgenocephalometric study*. Acta Odontologica Scandinavica. 1982; 40(3): 135-44.
22. Witkop CJ, Sauk JJ. *Hereditary enamel defects. In: Oral Facial Genetics*. Stewart RE, Prescott GH. Eds. P. 151. CV Mosby, St. Louis 1976.
23. Seow WK. *Enamel hypoplasia in the primary dentition: a review*. ASDC Journal of Dentistry For Children. 1991; 58: 441-452.
24. Lubinsky M, Angle C, Marsh, Pw, Witkop CJJr: *Syndrome of amelogenesis imperfecta, nephrocalcinosis, impaired renal concentration and possible abnormality of calcium metabolism*. American Journal of Medical Genetics. 1985; 20:233-43.
25. Lagerstrom M, Dahl N, Nakahori Y, Nakagome Y, Bäckman B, Landegren U, Pettersson U. *A deletion in the amelogenin gene (AMG) causes X-linked amelogenesis imperfecta (AIH1)*. Genomics. 1991; 10(4): 971-5.
26. Lau EC, Slavkin HC, Snead ML. *Analysis of human enamel genes: insights into genetic disorders of enamel*. Cleft Palate Journal. 1990; 27(2): 121-30.
27. Salido EC, Yen PH, Koprivnikar K, Yu LC, Shapiro LJ. *The human enamel protein gene amelogenin is expressed from both the X and the Y-chromosomes*. American Journal of Human Genetics. 1992; 50(2): 303-16.
28. Wright JT, Butler WT. *Alteration of enamel proteins in hypomaturation amelogenesis imperfecta*. Journal of Dental Research. 1989; 68(9): 1328-30.
29. Seow WK. *Clinical diagnosis and management strategies of amelogenesis imperfecta variants*. Pediatric Dentistry. 1993; 15(6): 384-93.
30. Largerström M, Dahl N, Iselius L, Bäckman B, Pettersson U. *Mapping of the gene for X-linked amelogenesis imperfecta by linkage analysis*. American Journal of Human Genetics. 1990; 46:120-25.
31. Lau EC, Mohandas TK, Shapiro LJ, Slavkin HC, Snead ML. *Human and mouse amelogenin gene loci are on the sex chromosomes*. Genomics. 1989; 4(2): 162-8.
32. Snead ML, Lau EC, Fincham AG, Zeichner-David M, Davis C, Slavkin HC. *Of mice and men: anatomy of the amelogenin gene*. Connective Tissue Research.1989; 22(1-4): 101-9.
33. Glimcher MJ, Friberg UA, Levine PT. *The isolation and amino acid composition of the enamel proteins of erupted bovine teeth*. Biochemical Journal. 1964; 93:202-10.
34. Weidmann SM, Eyre DR. *Amino acid composition of enamel protein in the fully developed human tooth*. Caries Research. 1967; 1(4): 349-55.
35. Weatherell JA, Weidmann SM, Eyre DR. *Histological appearance and chemical composition of enamel proteins from mature human molars*. Caries Research. 1968;2(4): 281-93.
36. Robinson C, Briggs HD, Kirkham J, Atkinson PJ. *Changes in the protein components of rat incisor enamel during tooth development*. Archives of Oral Biology. 1983; 28(11): 993-1000.
37. Wright JT, Robinson C, Kirkham J. *Enamel protein in smooth hypoplastic amelogenesis imperfecta*. Pediatric Dentistry. 1992; 14(5): 331-7.
38. Wright JT, Aldred MJ, Crawford PJ, Kirkham J, Robinson C. *Enamel ultrastructure and protein content in X-linked amelogenesis imperfecta*. Oral Surgery, Oral Medicine, Oral Pathology. 1993; 76(2): 192-9.
39. Seow WK. *Taurodontism of the mandibular first permanent molar distinguishes between the Tricho-dento-osseous (TDO) syndrome and amelogenesis imperfecta*. Clinical Genetics. 1993; 43(5.: 240-6)
40. Witkop CJJr. *Partial expression of sex-linked recessive amelogenesis imperfecta in females compatible with the Lyon hypothesis*. Oral Surgery, Oral Medicine, Oral Pathology. 1967; 23(2): 174-82.
41. Bäckman MD, Singer A. *Demonstration of the lyon hypothesis in X-linked dominant hypoplastic amelogenesis imperfecta*. Birth Defects: Original Article Series. 1971; 7(7): 204-9.
42. Simonsen RJ, Kanca J. *Surface hardness of posterior composite resins using supplemental polymerisation after simulated occlusal adjustment*. Quintessence International. 1986; 17(10): 631-3.
43. Wright JT. *Analysis of kindred with amelogenesis imperfecta*. Journal of Oral Pathology. 1985; 14(5): 366-74.
44. Bäckman B, Anneroth G. *Microradiographic study of amelogenesis imperfecta*. Scandinavian Journal of Dental Research. 1989; 97(4): 316-29.
45. Bäckman B, Anneroth G, Horstedt P. *Amelogenesis imperfecta: a scanning electron microscopic and microradiographic study*. Journal of Oral Pathology & Medicine. 1989; 18(3): 140-5.
46. Li RW. *Adhesive solutions: report of a case using multiple adhesive techniques in the management of enamel hypoplasia*. Dental Update. 1999; 26(7): 277-82, 284, 287-7.
47. Mahoney EK. *The treatment of localised hypoplastic and hypomineralised defects in first permanent molars*. New Zealand Dental Journal. 2001; 97(429): 101-5.
48. Rosenthaler H, Randel H. *Rotary reduction, enamel microabrasion, and dental bleaching for tooth color improvement*. Compendium of Continuing Education in Dentistry. 1998; 19(1): 62-7.
49. Heymann HO. *Conservative concepts for achieving anterior esthetics*. Journal of the California Dental Association. 1997; 25(6): 437-43.
50. Croll TP. *Enamel microabrasion for removal of superficial dysmineralisation and decalcification defects*. Journal of the American Dental Association. 1990; 120(4): 411-5.
51. Andlaw RJ. *The treatment of hypoplastic and hypomineralised teeth*. Proceedings of the British Paedodontic Society. 1983; 13:25-30.
52. Dunne SM, Gainsford ID, Wilson NH. *Current materials and techniques for direct restorations in posterior teeth. Part 1: Silver amalgam*. International Dental Journal. 1997; 47(3): 123-36.
53. Harley KE, Ibbetson RJ. *Anterior veneers for the adolescent patient: 2. Porcelain veneers and conclusions*. Dental Update. 1991; 18(3): 112-6.
54. Rada RE, Hasiakos PS. *Current treatment modalities in the conservative restoration of amelogenesis imperfecta: a case report*. Quintessence International. 1990;21(12): 937-42.
55. Venezie RD, Vadiakas G, Christensen JR, Wright JT. *Enamel pre-treatment with sodium hypochlorite to enhance bonding in hypocalcified amelogenesis imperfecta: case report and SEM analysis*. Pediatric Dentistry. 1994;16(6): 433-6.
56. Croll TP, Castaldi CR. *The preformed stainless steel crown for restoration of permanent posterior teeth in special cases*. Journal of the American Dental Association. 1978; 97(4): 644-9.
57. Croll TP. *Permanent molar stainless steel crown restoration*. Quintessence International. 1987;18(5): 313-21.
58. Perez B, Yakir O, Fuks AB. *Follow up after root canal treatment of young permanent molars*. Journal of Clinical Pediatric Dentistry. 1997; 21(3): 237-40.
59. Mackie IC, Blinkhorn AS, Davies PHJ. *The extraction of permanent first molars during the mixed-dentition period – a guide to treatment planning*. Journal of Paediatric Dentistry 1989;5:85-92.
60. Penchas J, Peretz B, Becker A. *The dilemma of treating severely decayed first permanent molars in children: to restore or to extract*. Journal of Dentistry for Children. 1994; 61(3): 199-205.
61. Hashimoto M. *Effects of Nd: YAG laser irradiation on acid resistance of defective rat enamel*. Japanese Journal of Pedodontics. 1990;28(4): 956-67.
62. Rosenblum SH. *Restorative and orthodontic treatment of an adolescent patient with amelogenesis imperfecta*. Pediatric Dentistry. 1999;21(4): 289-92.
63. Rada RE, Hasiakos PS. *Current treatment modalities in the conservative restoration of amelogenesis imperfecta: a case report*. Quintessence International. 1990;21(12): 937-42.
64. Greenfield R. Iacono V. Zove S. Baer P. *Periodontal and prosthodontic treatment of amelogenesis imperfecta: a clinical report*. Journal of Prosthetic Dentistry. 1992; 68(4): 572-4.
65. Lumley PJ, Rollings AJ. *Amelogenesis imperfecta: a method of reconstruction*. Dental Update. 1993; 20(6): 252-5.
66. Bedi R. *The management of children with amelogenesis imperfecta*. Restorative Dentistry. 1989; 5(2): 28, 31-4.
67. Mackie IC, Blinkhorn AS. *Amelogenesis imperfecta: early interception to prevent attrition*. Dental Update. 1991; 18(2): 79-80.
68. Wright JT, Waite P, Mueninghoff L, Sarver DM. *The multidisciplinary approach managing*

enamel defects. Journal of the American Dental Association.1991;122(2): 62-5.

69. Bouvier D, Duprez JP, Bois D. *Rehabilitation of young patients with amelogenesis imperfecta: a report of two cases*. Journal of Dentistry for Children. 1996; 63(6): 443-7.
70. Alexander SA. *The treatment of hypocalcified amelogenesis imperfecta in a young adolescent*. Journal of Pedodontics. 1984; 9(1): 95-100.
71. Rada RE, Hasiakos PS. *Current treatment modalities in the conservative restoration of amelogenesis imperfecta: a case report*. Quintessence International. 1990; 21(12): 937-42.
72. Gibbard PD. *The management of children and adolescents suffering from amelogenesis imperfecta and dentinogenesis imperfecta*. International Journal of Orthodontics. 1974; 12(4): 15-25.
73. Bäckman B. *Amelogenesis imperfecta--clinical manifestations in 51 families in a northern Swedish county*. Scandinavian Journal of Dental Research. 1988; 96(6): 505-16.
74. Rowley R, Hill FJ, Winter GB. *An investigation of the association between anterior open-bite and amelogenesis imperfecta*. American Journal of Orthodontics.1982; 81(3): 229-35.
75. Nakahori Y, Takenaka O, Nakagome Y. *A human X-Y homologous region encodes 'amelogenin'*. Genomics. 1991; 9(2): 264-9.
76. Aldred MJ, Crawford PJ, Roberts E, Gillespie CM, Thomas NS, Fenton I, Sandkuijl LA, Harper PS. *Genetic heterogeneity in X-linked amelogenesis imperfecta*. Genomics. 1992; 14(3): 567-73.
77. Seow WK, Latham SC. *The spectrum of dental manifestations in vitamin D-resistant rickets: implications for management*. Pediatric Dentistry. 1986;8(3): 245-50.
78. Johnson A, Winstanley RB. *Use of simple overdentures in the treatment of young patients with developmental anomalies*. Quintessence of Dental Technology. 1987; 11(1): 27-33.
79. Renner RP, Ferguson FS. *Overdenture management of amelogenesis imperfecta*. Quintessence International. 1983; 14(10): 1009-22.
80. McLarty EL, Giansanti JS, Hibbard ED. *X-linked hypomaturation type of amelogenesis imperfecta exhibiting lyonisation in affected females*. Oral Surgery, Oral Medicine, Oral Pathology. 1973; 36(5): 678-85.

ANZSPD Federal Report

The ANZSPD Federal Council met in Sydney in September 2003. As usual, a range of topics was covered.

1. The Society is taking advantage of a kind offer by the Australian Dental Association to assist affiliated societies in establishing websites. John Winters has kindly agreed to continue to liaise with Ron Robinson, to see this project through to conclusion.

2. A comprehensive report on the 13th ANZSPD Federal Convention in Brisbane in November 2002 was presented. The success of the meeting was acknowledged by the Council, with well deserved congratulations to the Queensland Branch Local Organising Committee. This success was also financial – a total profit of AU$13,463.00 (after GST) was realised. This profit is shared between the Federal body and the Queensland Branch. The importance of the appointment of a professional conference organising company was noted, especially as these Conventions are becoming 'major productions'.

3. The next Australian Dental Association Congress is to be held in Adelaide 4 – 8 March 2005. It is hoped that the scientific programme will have a paediatric dental component, and to this end, ANZSPD will be making representations (principally through our South Australian Branch) to the Congress Local Organising Committee.

4. A good deal of time was devoted to fine tuning the Colgate Post-graduate Research Project Competition rules. This competition has become a very important part of the programme of each ANZSPD Convention, and with the greater number of post-graduate students in Australia and New Zealand now, this fine tuning was always going to be necessary. The notices for this competition for the Convention in Melbourne in March are about to be sent out to Course Controllers.

5. R.K. Hall Visiting Lecturer. The 2003 tour of Associate Professor Kim Seow has now been completed, with visits to Palmerston North and Dunedin in New Zealand and Hobart in Tasmania. The R.K. Hall tour is usually held in the year following the ANZSPD Convention. The Federal Council decided the next tour should be delayed until February – March 2006, quite obviously because of the busy 2005, with both the Adelaide ADA Congress and the IAPD Congress in Sydney in that year. The tentative plan was for the chosen lecturer to visit South Australia, Queensland and the Australian Capital Territory. It is hoped a lecturer will be appointed in the next couple of months.

6. The 15th ANZSPD Convention. The Western Australian Branch has undertaken to conduct this meeting. In a rather bold departure from tradition, the Branch has proposed the meeting be held in the town of Broome, the Kimberley resort in the far north of the state, probably in August 2006. Watch this space!

7. The ANZSPD Essay Competitions topics for 2004 were chosen. The post-graduate topic is: 'Enamel Hypoplasia in First Permanent Molar Teeth. Discuss the aetiology, prevention and management of this condition'. The under-graduate essay topic is: 'Discuss the diagnosis, classification and management strategies of Ectodermal Dysplasia'.

Another matter which had been raised at the General Meeting of the Society in Brisbane in November 2002, and which was raised again at the Council meeting in Sydney, relates to ANZSPD membership and communication between provincial branches and the federal body. It is probably appropriate to run through how this all operates, in view of the obvious confusion which exists in the minds of many members and branch officers.

- When a member joins a provincial branch, they can or will become a member of the federal body.

- With the federal body, there are five categories of membership – full members; full members who choose to take individual membership of the International Association of Paediatric Dentistry through ANZSPD; associate members; associate members who choose to take individual membership of the International Association of Paediatric Dentistry through ANZSPD; and Honorary Life Members.

- Some full members choose to take out their individual membership of the International Association of Paediatric Dentistry themselves by dealing directly as an individual with the IAPD Secretariat.

Continued on page 23...

ANZSPD – Branch News 2003

New South Wales

The highlight so far in 2003 for the NSW branch of ANZSPD has been a one day conference on 27th of June. The day involved a series of half hour lectures with the theme and title of the day being 'Management strategies of the difficult patient'. Over 100 general dentists and specialists attended this meeting at our regular meeting venue- the Duxton Hotel, North Sydney. The speakers for the day were from a wide range of specialty groups including Endodontist, Orthodontist, Dental Assistants, Clown Doctors, Medical Practitioners, Lawyers and Special Care Dentists. Dr's Peter Wong and Angus Cameron represented the Paediatric Dentists by delivering interesting and well received lectures on management of difficult patients in private and public paediatric dental practice respectively. Great support for this day was provided by our Primary Sponsor Colgate. Other sponsors were GC, Medfin, Guild Insurance, Air Liquide and Halas.

Although this day has taken up the majority of the NSW committee's time there have been two other successful meetings. Dr Sally Hibbert who is the newest Consultant at Westmead Centre for Oral Health, spoke on 'Why do we restore primary teeth?' and Ms Ms Sue Larkey presented on the 'Autism Spectrum'. Dr Michael Malandris, a post graduate in Paediatric Dentistry at the University of Sydney also spoke at our last meeting on the 'Treatment of posterior cross bites in the primary dentition'. Our last meeting of the year will be in November when we are pleased to have Dr Kerrod Hallett from Queensland as our invited speaker.

Plans for 2004 include three dinner meetings and the continuation of planning for the International Association of Paediatric Dentistry Meeting in Sydney, 2005. Plans are well underway to make this meeting a showcase of Australia and New Zealand Dentistry as well as continuing to promote oral health for all children.

With the end of the year approaching fast all members of NSW ANZSPD are anticipating next year's biannual conference in Melbourne in March. We are hoping for the reinstatement of 'Wongy's Bar' or possibly 'Olsen's Bar'?. See you all there.

Erin Mahoney

The organising committee for the ANZSPD 'Management of Difficult Patient' with Dr Fruit Loop, Clown Doctor, who was one of the invited speakers at this successful one day meeting

Victoria

The Victorian Branch 2003 educational programme has been well attended at all four dinner meetings held so far this year at University House.

At the first meeting for the year in February, members were provided with 'The orthodontist's challenge – what to do with those unusual cases' by Dr Brittany Shearn. Drs Karen Kan and Chris Olsen also provided a segment on their experiences in teaching paediatric dentistry at Cambodia's only dental school in Phnom Penh for a short period in 2002.

Our April meeting was addressed by Dr Peter King, who gave an interesting presentation on the 'Unique oral health problems of people with disabilities', drawing on his extensive experience and knowledge in this area. Fittingly at this meeting the Dr Des Crack Memorial prize was awarded to new graduate, Dr Yvonne Yu-Ying Chang, who showed the greatest interest and ability in the field of paediatric dentistry in final year 2002. Dr Karmun Chan, Senior Registrar at the Royal Children's Hospital of Melbourne also presented an informative case report on Cleidocranial Dysplasia.

In June, Dr Ivan Darby, Senior Lecturer in Periodontology, treated members to an entertaining update entitled 'Bugs, Who needs them to get disease'. This was preceded by a case report on histiocytosis and its oral manifestations by paediatric dentistry postgraduate, Dr Yaso Ramadas.

The August meeting was addressed on the topic 'Is your patient allergic to you? Potential implications for the dental practitioner', by Dr Jo Smart, Paediatric Allergist at the Royal Children's Hospital of Melbourne. Dr Smart provided information on allergies in dentistry, including to antibiotics and latex, as well as on oral allergy syndrome, allergic reactions to local anaesthetics and oro-facial granulomatosis. Paediatric dentistry postgraduate, Dr Feda Zawaideh, also gave an interesting case presentation on Noonan's Syndrome.

Dr Simon Wylie, Prosthodontist, is to give the Elsdon Storey Memorial Lecture in October, entitled 'Prosthodontics and generation 'F' – Does the fluoride generation need us? Treatment Planning Adolescents Today for Tomorrow'. This concludes our Scientific programme for 2003, however our End of Year Christmas function is to be held on Friday 5th December.

Chris Olsen

Tasmania

The R.K. Hall Visiting Guest Lecturer Series was held in Hobart over the AFL Grand Final weekend. Normally this would have affected the attendance but it appears one can attend a course presented by an excellent guest speaker (Dr Kim Seow) and still keep tabs on the score during the interval.

Dr Seow presented an excellent day's worth of visual and scientific information which kept the audience stimulated throughout, definitely 'New Thoughts for Old'.

Also in attendance was Dr. Chris Olsen, who presented on Traumatology, and if I may add a mighty fine igloo was included.

The audience came from across Tasmania, many having driven for several hours to attend. It was fantastic to see so many therapists included, and the branch hopes to see our membership swell this year to allow the free flow of many more guest speakers to Tasmania.

Thank you all for your enthusiasm and attendance. It is much appreciated.

Tasha Dodd

Western Ausralia

The WA Branch has, once again, held a very successful mid-winter meeting. On the last weekend of July, members and partners travelled to the wheatbelt town of York. It is 100 kms east of Perth. York is an historic town, having been first settled in 1831, only two years after the establishment of the colony of Western Australia in Fremantle and Perth. The venue for the ANZSPD meeting, Faversham House, has been there for almost all of that time. It was originally built in 1849, and it has been meticulously restored in the last five years. It certainly is a grand house.

Our guest of honour for this meeting was ANZSPD Federal President, Dr Chris Olsen. The meeting started after lunch on Saturday, with Chris presenting on a number of topics – Managing Bumps and Knocks, Guided Tissue Regeneration in Managing Supernumeraries, Interventional Orthodontics and finally, Taking Paediatric Dentistry to Cambodia. It was a fascinating and varied collection of items. The evening saw a quite magnificent dinner in the impressive dining room of the house, before members and partners retired to the lounge room for what could be described as a characteristic ANZSPD conclusion to a terrific evening! A great time (and late time for some) was had by all.

Sunday morning saw the other regular feature of these mid-winter meetings, the Pot Pourri. As usual, there was a remarkable variety of presentations by members, ranging from orthodontics, to trauma, to prevention in practice, to restoration of primary teeth, to possible fusion of erupting permanent incisors, and also as usual, there was more material than time available. Chris Olsen commented favourably on this format, and suggested it was a formula he would be suggesting the Victorian Branch might like to try. Based on the success of the format in WA over many years, it would be a suggestion well worth taking up.

The next meeting for the Branch will be the visit of Dr Sally Hibbert to Perth. She will present on the topic of 'Why Save Primary Teeth?' The support speaker will be Perth orthodontist, Dr Peter Dillon, who will speak on 'Space Maintenance – Where, Why, When and How.' The branch will hold its Annual General Meeting at the conclusion of this course.

It had been planned to hold a combined ANZSPD and Australian Society of Endodontology day course on Dental Trauma in November this year. This meeting has now been postponed until May 2004. It will be a busy start to 2004 – in addition to the Dental Trauma day, IAPD Secretary Professor Gerry Wright has agreed to accept the University of Western Australia A.J. Herman Fellowship for 2004 and will spend two weeks in Perth in early March. His visit will be very welcome. He will deliver lectures to undergraduates, consult in undergraduate teaching clinics and participate in clinics at the Princess Margaret Hospital for Children.

Alistair Devlin

New Zealand

The NZ branch were very pleased with the response to Dr Kim Seow's tour 'New Thoughts for Old' as part of the R.K. Hall 2003 lecture series. Her considerable research skills and sympathetic approach to early childhood caries in particular was appreciated by all who heard her (about 270 in all).

The NZ executive have also been involved in submissions to the (NZ) Accident Compensation Commission which is considering increasing the publicly available funds for dental trauma management for under 18 year olds. We pointed out that deciduous teeth can also be traumatised and the need for regular reviews of trauma as set out by Andreasen et al. in "Guidelines for the Evaluation and Management of Traumatic Dental Injuries; Dent Traumatol, 2001. The inclusion of more recent interventions such as bonding of tooth fragments was also supported. Our parent body NZDA had also provided significant input to this ACC process independently, which we fully endorsed.

The annual hospital dental conference saw several excellent presentations by members of the society with Dr Bernadette Drummond (review of care undertaken under GA in Dunedin) and Dr Erin Mahoney (on restorative choices for deciduous teeth) both achieving praise for their presentations.

At the time of writing Dr Katie Ayers is expecting to deliver her 2nd baby.

We are all looking forward to Melbourne in 2004.

MaryAnne Costelloe

Hang up your highspeeds – it's time for a holiday

The fate of carious deciduous teeth in the United Kingdom

A literature critique by David J Manton
School of Dental Science, University of Melbourne

Recently, several articles have been published in the British Dental Journal that investigate the outcome of different treatment modalities for deciduous teeth in UK children.[1,2,3] The main conclusion of these articles has been that there is very little difference in long term outcome between those teeth that have been restored and those teeth that have not been restored. Not surprisingly, this has raised issues regarding the effectiveness of public funds allocated to paediatric dentistry and the appropriateness of the treatment provided to the child population by general dentists in the UK. In the light of this, public dental authorities are now asking similar questions in Australia.

A study by Levine et al of children treated from 1976 until 1996 in Northern England by a general dental practitioner retrospectively reviewed the clinical treatment records of child patients of two dental clinics.[1] The objective was to review the outcome of treatment/non treatment of carious lesions in primary molars. The study was limited to those children who attended the clinic annually (after initial examination) for the life of their deciduous dentition. The most likely causes of pain were carious molars with pulp involvement diagnosed before three years of age. Of the total number of carious teeth, 74 per cent had exfoliated without pain. The conclusion of this study was that the majority of carious deciduous teeth exfoliated without symptoms. This still leaves approximately one in five teeth that required extraction.

Some inadequacies in the design of this study are apparent. There was a lack of examination standards or criteria. A tooth with 'carious pulp involvement' was recorded but no clinical criteria were described or confirmatory radiographs routinely taken, with only visual determination of a probable carious exposure being used. The recording of pain was based on presenting pain, with no obvious effort to elicit any verbal pain or infection history. The diagnostic criteria used for infection were based on the oral findings at presentation, with no criterion for radiographic diagnosis. Teeth were eliminated from the study if they had been restored whilst being symptom free, with the study therefore biased towards the failure of restorations. The method of treatment for the restoration group is unknown, especially the criteria used to determine if pulp therapy was required.

Another area of concern within the article is that the design limits itself to regular attendees at the same clinics. This possibly eliminated a large number of potential subjects who were symptomatic attendees or those who had needed to seek emergency care elsewhere. It is a non-representative sample biased towards those children and parents who have sought or required ongoing dental care at the same clinic.

A second recent article by Tickle et al (2002) followed a similar theme to the first. The authors investigated the survival of 4,056 teeth recorded from December 1990 to December 1999 as carious, or having had some restorative intervention by 50 general dentists in the North West UK. Of the 4,056 teeth, 1,789 (44.1%) were extracted.[2] Not all of these teeth were extracted due to pain. Among the first deciduous molars that had never been restored, 23 per cent were extracted due to pain or sepsis, and the same fate awaited 20 per cent of the restored teeth. There was little difference in the extraction rate between those that had been restored, and those left carious. In this same cohort, almost half (48%) of the children had experienced at least one episode of dental pain, with the prevalence of pain directly related to the total decay experience of the child.[3]

General issues

The implications of leaving the cause of a potentially serious dental infection untreated are concerning. Apart from the pain and suffering that may occur, there are possible legal consequences from the lack of, or inadequate, treatment of disease. The legal issues include the standard of care (treating to a recognised professional standard including codified practice procedures), contributory negligence (to act or not act in a manner that contributes to injury), and reasonable foreseeability (to be able to foresee the consequences of acting or not acting). These all appear to have a possible relationship to treating/not treating the disease and informing the guardian of the possible consequences of treating / not treating.

The health effects of the symptomatic treatment of chronic dental infection with antimicrobials are not considered. The long and short term benefits and success of appropriate early restorative and endodontic intervention when providing paediatric dental care have been ignored in both these studies, and replaced by 'extraction when painful; leave until ache' approach. Reported pain and suffering seem to be accepted by the authors as an unavoidable consequence of childhood, whilst research has indicated stainless steel crowns and pulpotomies provide excellent long term restorative solutions for primary molar teeth.[4] The comparative survival rate of stainless steel crown restorations has been reported to be greater than that of amalgam restorations, with amalgams more than twice as likely to fail than SSC.[5-7]

The need for 20 – 44 per cent of carious or restored teeth to be extracted is concerning, as are the long term behavioural consequences of this treatment modality.[1,2] There is no indication of the dmft of the study groups, although the regional caries prevalence in the first study is 'higher than the national average'.[1] The number of missing (due to caries) deciduous teeth in Australian children

in 1998 ranged from 0.02 to 0.08 in the age group of four – twelve years.[8] When this is considered with regard to the dmft range in this study (0.43 – 1.81), extraction would represent only a tiny proportion of the dental experience of Australian children.[8] The comparison with the figures of Levine et al, although not direct, where one in five carious deciduous molars were extracted, seems heavily weighted towards extraction.[1,8] The orthodontic effects due to tooth movement after tooth loss or tooth structure loss of the deciduous molars are also ignored.

One important point that is not mentioned is whether parents/ guardians were properly informed of the different types of treatment available, the relevant consequences of these, and whether the parent had any input into treatment type undertaken. This apparent lack of informed consent may only reflect the prevailing attitudes in the era during which the treatment was performed. This raises legal issues concerning consent, as the parent/guardian may have been given no input into what may be considered sub-optimal treatment.

Discussing the fate of carious deciduous teeth, Duggal in 2002 has raised the issue that in the UK, the poor outcome of primary molar restorations may be due to poor diagnosis and technique.[9] The appropriate and timely treatment of the deciduous pulp and subsequent restoration of the tooth is the issue, not whether the tooth should be restored or left untreated.[10]

The manner in which children's dentistry is funded may also influence the dental treatment of children. In the UK, the majority of paediatric dentistry is funded by the government and provided by general dental practitioners.[11] Payment is made partly by a capitation fee and partly by fee per item. This arrangement of funding may bias the treatment towards quick 'cost effective' methods rather than the more time consuming complete restorative treatment previously mentioned. There are also community based clinics (formerly 'school dental clinics') similar to those in Australia with salaried dentists. Very few children have private dental care at present in the UK.[11]

In summary, these articles indicate that the treatment modalities currently used in the UK under the General Dental Service require review. It appears that dental treatment received by a large number of children is inappropriate and provides little advantage over receiving no treatment at all. In all likelihood, this is due to the lack of appropriate diagnosis and treatment planning, ignoring the fact that the majority of deciduous molar teeth have advanced pulpal inflammation once the marginal ridge is breached. Treatment that does not take this into account will in all likelihood fail.[10]

The conclusions of the UK articles discussed should not be used as a justification for treatment being limited to the symptomatic removal of deciduous teeth; rather, it indicates a need for the review of paediatric dentistry performed by general dentists in the UK.[1,2,3] The findings are 'simply a reflection of an under-funded, under-skilled and inadequate approach to paediatric dentistry within the General Dental Service of the NHS'.[12]

Similar conclusions may be drawn for the situation that currently occurs in Australia. In a climate of limited funding for public dental health services, methods of cost cutting or saving are constantly sought. Any move towards a similar treatment philosophy to that portrayed in the Levine et al article for paediatric dentistry will fail. Apart from the short term clinical consequences of inadequate and inappropriate dental treatment for children, the long term general health, behavioural and orthodontic outcomes may outweigh any cost saving made.

A similar study of treatment provided by private practitioners in Australia would provide an interesting comparison, especially due to the vastly different funding arrangements and the greater availability of preventive and restorative care provided through the public sector.

References

1. Levine RS et al. *The fate of 1587 unrestored carious teeth: a general practice based study for northern England.* Brit Dent J 2002; 193(2):99-103.
2. Tickle M. et al. *The fate of the carious primary teeth of children who regularly attend the general dental service.* Br Dent J. 2002;192(4):219-23.
3. Milsom KM et al. *Dental pain and dental treatment of young children attending the general dental service.* Br Dent J 2002; 192(5):280-84.
4. Roberts JF, Sherrif AM. *The fate and survival of amalgams and preformed crown molar restorations placed in specialist paediatric dental practice.* Br Dent J 1990; 169:237-44.
5. Levering NJ, Messer LB. *The durability of of primary molar restorations: I. Observations and predictions of success of amalgams.* Pediatr dent. 1988;10: 78-80.
6. Messer LB, Levering NJ. *The durability of primary molar restorations: II. Observations and predictions of success of stainless steel crowns.* Pediatr Dent. 1988;10:81-85.
7. Tate AR, Ng MW, Needleman HL, Acs G. *Failure rates of restorative procedures following dental rehabilitation under general anaesthesia.* Pediatr Dent. 2002;24:69-71.
8. Armfield JM, et al. 2001. *The child dental health survey, Australia 1998.* AIHW Cat. No. DEN88. Adelaide: Adelaide University (AIHW Dental Statistics and Research series No. 24).
9. Duggal MS. *Carious primary teeth: their fate in your hands.* Br Dent J 2002; 192: 215.
10. Duggal MS, Nooh A. *The relationship between extent of carious involvement of the marginal ridge and pulp inflammation of primary teeth.* J Dent Res 1999; 78:298.
11. Levine R. *Personal communication by email,* 18/02/2003.
12. Lennon MA. *Pain and treatment of carious primary teeth.* Br Dent J 2002; 192:272-73.

An *in vitro* investigation of the effects of casein phosphopeptide – stabilised amorphous calcium phosphate on

Erosion of human dental enamel by sports drinks

2002 Colgate Postgraduate Research Project Competition Winner

L Ramalingam[1], L B Messer[2,3], E C Reynolds[2]
School of Dental Science, University of Melbourne

[1] Formerly postgraduate student in Pediatric Dentistry, School of Dental Science, University of Melbourne, and honorary dental officer, Royal Children's Hospital, Melbourne

[2] School of Dental Science, University of Melbourne

[3] Corresponding author: Professor Louise B Messer, School of Dental Science, The University of Melbourne

Abstract

Sports drinks (eg. Powerade™) have been found to erode human dental enamel. This study determined a minimal concentration of casein phosphopeptide-stabilised amorphous calcium phosphate (CPP-ACP) added to Powerade™ that would eliminate erosion *in vitro*. Human enamel specimens immersed in Powerade™; Powerade™ plus 0.063%, 0.09%, 0.125% or 0.25% CPP-ACP; or DDW, were examined by stereomicroscopy, profilometry and SEM. With increasing CPP-ACP concentrations, the pH of solutions increased and titratable acidity decreased. Specimens in Powerade™ showed erosive steps (mean depth: 38.70 kA ± 5.60 SD), eliminated on adding CPP-ACP (0.09 – 0.25%) but not by 0.063% CPP-ACP. Untrained tasters could not differentiate Powerade™ from Powerade™ plus 0.125% CPP-ACP. Adding CPP-ACP significantly reduced the erosivity of Powerade™ and resulted in microscopic surface irregularities.

Introduction

Dental erosion is a chronic loss of hard tissue, chemically removed by acid and/or chelation without bacterial involvement (ten Cate and Imfeld, 1996). Acid sources include diet, medications and lifestyle (Zero, 1996). Erosion prevalence is increasing, reflecting wide availability and frequent consumption of acidic drinks (Nunn, 1996). Other dietary sources include vinegar, fruit juices, fruits and sports drinks (Jarvinen et al., 1991; Milosevic, 1997).

Sports drinks are formulated to be palatable, prevent dehydration, supply carbohydrates and replenish lost electrolytes (Coombes and Hamilton, 2000). Consumption by athletes, recreational users and adolescents has increased with promotional advertising (O'Dea and Rawstone 2000, Sirimaharaj et al. 2002, Mathew et al. 2002). The low pH, high titratable acidity and viscosity of sports drinks increase erosivity, and erosion is promoted by consumption during periods of dehydration and low salivary flow (Milosovic, 1997). Drink erosivity also depends on acid type and the concentrations of calcium, phosphate and fluoride (Lussi et al., 1995; Hughes et al., 2000).

The erosivity of manufactured products is modifiable, but complete removal of acids affects palatability and stability and this approach is rarely feasible (Grenby, 1996). Products may be modified by calcium and/or phosphate supplementation (Reynolds, 1998; Hughes et al., 1999a; Larsen and Nyvad, 1999; Hughes et al., 2000), but palatability and texture influence consumer acceptance (Grenby, 1996; Hughes et al., 2000). An *in vitro* study showed three sports drinks (Powerade™, Gatorade™ and Sports Plus™) are erosive; supplementing Powerade™ with the milk protein, casein phosphopeptide-stabilised amorphous calcium phosphate (2% CPP-ACP) with or without pH modification, eliminated erosion *in vitro* (Vasan, 1998).

In vitro studies lack erosion-protective factors found intraorally, and biological factors *in vivo* may reduce the erosivity of acidic drinks. These include consumption pattern (slow sipping, delayed swallowing, straw sipping), salivary flow and buffering, pellicle, tooth composition, and orofacial musculature (Zero, 1996; Imfeld, 1996; Hall et al., 1999; Hunter et al., 2000; Hughes et al., 2000). Thus *in vitro* studies may overestimate hard tissue removed by erosion.

This *in vitro* study extended the work of Vasan (1998), confirming preliminary findings and determining a minimal concentration of CPP-ACP added to Powerade™ that could prevent enamel erosion. Surface changes were recorded and test solutions were studied for pH, titratable acidity and taste.

Materials and methods

Specimen preparation

Fifteen human erupted third molars, caries-free and defect-free, were stored in thymol solution. Transverse, bucco-lingual segments (2 mm thick) were cut mid-coronally using an internal annulus saw microtome (Leitz 1600, Ernst Leitz, Wetzlar, Germany) under water irrigation and sectioned mesiodistally, providing two specimens per tooth. Specimens were divided randomly into six groups (five per group), embedded in epoxy resin (EPOFI Epofix, Radiometer, Copenhagen, Denmark) in plastic moulds (1x1x1.5cm), then machine-polished (Struers, Rotopol-21, Copenhagen, Denmark) with silicon carbide paper (grades 600-1200) under water irrigation, removing 50-100 µm to produce flat enamel surfaces. Control areas were painted with fingernail varnish, exposing a 1mm test window demarcated by scalpel cuts.

Solution preparation

Solutions were prepared from commercial berry-flavored Powerade™ (P, Coca-Cola Co, Sydney, Australia), with CPP-ACP (batch no. 850143, Bonlac, Melbourne, Australia) added at concentrations of 0.063%, 0.09%, 0,125% and 0.25%. The control was DDW. To prepare test solutions, 0.5g of CPP-ACP powder was added to 200ml P and serially diluted. Vasan (1998) found P contains calcium (36.3 µg/mL), phosphate (13.15 µg/mL) and fluoride (24.16 µg/mL).

Experimental procedure

A 50ml aliquot of each solution was agitated (water incubator) at 37°C for five minutes. The resin blocks were added, agitated for 30 minutes, then rinsed in DDW and the varnish removed with acetone on a soft cloth.

Enamel surfaces were examined by stereomicroscopy (20X), then profiled (Alpha-Step 250 Tencor™ surface profilometer, Tencor Co, Michigan, USA). The profilometer stylus is a 60-degree cone rounded to a spherical tip (0.2 micron radius) which profiles the surface (5.0 mg tracking force). Each specimen was profiled three times (total 15 profiles per group); erosion depth was recorded at the deepest point. Specimens were then gold sputter-coated (Edwards, S150B, West Sussex, England) for SEM (XL 30 FEG, Philips, The Netherlands). Etched enamel surfaces were classified (Silverstone et al., 1975) and digital images were viewed using Corel Photo Paint 8™ (version 8.0, Corel Corp, Austin, Texas, USA).

Measurements of solutions

The pH and titratable acidity (PHM 84 Research pH meter™, TTT 80 Titrator™, ABU 80 Autoburette™, Radiometer, Copenhagen, Denmark) were measured in triplicate for three aliquots per solution. For titratable acidity, the volume of freshly prepared 0.1M potassium hydroxide required to raise the pH of a 5ml aliquot to 7.00 was measured at 37°C.

Taste test

A duo-trio test (Meilgaard et al., 1991) assessed if untrained tasters (20 staff volunteers) could differentiate P from P plus 0.125% CPP-ACP at room temperature. Both samples were used randomly as reference samples. Each taster tasted the reference sample and then two coded samples (one matched the reference sample), rinsing with tap water between samples. The taster identified which coded sample matched the reference sample; if uncertain, he/she indicated the answer was guessed.

Statistical analysis

Data were entered into SPSS (version 10.0.5) for WindowsTM and Excel™ (SPSS Inc, Chicago, USA). Mean values for pH and titratable acidity ($\alpha = 0.01$, adjusted for Bonferroni's correction) and erosion depths ($\alpha = 0.05$) were compared (one-way ANOVA). The minimum number of correct answers for 20 tasters in the taste test is 15 (15 or greater rejects the assumption of "no difference" between samples; $\alpha = 0.05$) (Meilgaard et al., 1991).

Results

Test solutions

Post-test, all solutions of P with or without CPP-ACP remained clear red and precipitate-free. With increasing CPP-ACP concentrations, the pH increased (from 2.709 for P to 3.903 for P plus 0.25% CPP-ACP), and the titratable acidity (TA) of solutions decreased. All pH values were significantly lower than for DDW ($p < 0.01$) and the TA of P plus 0.125% or 0.25% CPP-ACP was significantly lower than for P ($p < 0.01$).

Stereomicroscopy

All specimens in P with or without CPP-ACP stained red; those in P or P plus 0.063% CPP-ACP appeared etched with a loss of enamel surface gloss. Specimens in P plus 0.09% or 0.125% CPP-ACP showed less etching with little loss of gloss; those in P plus 0.25% CPP-ACP were unetched and glossy. Specimens in DDW were unstained, unetched and glossy.

Surface profilometry

The mean erosion depth (38.70 ± 5.60 kA) of specimens in P significantly exceeded that of all specimens in other test solutions and DDW ($p < 0.05$), showing a sharp step and convex floor. Specimens in P plus 0.063% CPP-ACP showed a shallower step (17.98 ± 3.05 kA), but mean depths did not differ significantly from those in P ($p > 0.05$). Specimens in P plus CPP-ACP at concentrations of 0.09%, 0.125%, 0.25%, or DDW, did not show erosion steps and mean erosion depths did not differ significantly from each other ($p > 0.05$), but all values differed significantly ($p < 0.05$) from mean erosion depths of specimens in P with and without 0.063% CPP-ACP.

Scanning electron microscopy

Specimens in P showed erosion steps between test and control areas (250X), Type 2 etching, and scattered areas of Type 3 (porous enamel). The enamel prism cores appeared intact with etched peripheries. Polishing lines were present on control areas only. At 1,000X, the inter-rod areas were porous. At 4,000X irregular etching was noted with areas of differentially etched enamel, and undercut regions (not profiled by the stylus) between test and control areas were apparent.

Specimens in P plus 0.063% CPP-ACP showed indistinct erosion steps gradually progressing from control to test areas. Areas of unaffected and etched enamel were interspersed; the enamel prism orientation was unclear (600X). No particular etch pattern was observed and the surface was highly irregular (1,000X); at 4,000X, test areas showed superficial rod-shaped particles.

Specimens in P plus 0.09% CPP-ACP showed polishing lines on control and test areas without erosion steps. At 150-600X, superficial irregularities including porosities were noted on test enamel, without an identifiable etch pattern. At 4000X, the surface was irregular and finely amorphous, including globular areas and unidentifiable enamel prisms.

Specimens in P plus 0.125% CPP-ACP showed polishing lines on control and test areas but no erosion steps (150X). Discrete areas of superficial surface irregularities occurred at the junction of control and test enamel (600X). At 1,200X, the enamel cores were etched in isolated areas and surface irregularities were distributed over all test enamel. At 4,000X, the irregularities appeared globular.

Specimens in P plus 0.25% CPP-ACP had few irregularities on test enamel (150X). Polishing lines were seen and enamel etching was noted in isolated areas (600X, 1,200X). Specimens in DDW showed smooth surfaces with

polishing lines, without erosion steps or superficial irregularities (100X).

Taste test

Fourteen tasters responded without guessing (ten correct, four incorrect), and six guessed responses (three correct, three incorrect). Correct and incorrect answers for each reference solution were distributed equally, indicating no bias. Considering guesses as incorrect, ten tasters matched samples correctly. Since at least 15 correct identifications are required to conclude statistical significance (Meilgaard et al., 1991), any taste difference between P with and without 0.125% CPP-ACP was considered undetectable.

Discussion

This *in vitro* study showed that enamel erosion by Powerade™ could be eliminated by adding 0.09-0.25% CPP-ACP. The lack of standardised techniques to determine the erosivity of drinks complicates comparisons between studies. Affecting dissolution, variables include enamel source (human or bovine, deciduous or permanent, powdered hydroxyapatite), temperature, specimen agitation, exposure time and volume, and evaluation methods (Zero, 1996; Larsen and Nyvad, 1999). The present study used mid-coronal buccal/lingual surfaces for standardization. Surfaces can vary in erosion susceptibility; prismatic enamel is more susceptible than aprismatic enamel which erodes irregularly (Meurman and Frank 1991; ten Cate and Imfeld, 1996). Aprismatic enamel occurs on all third molars, averaging 16-45 µm on buccal and lingual surfaces (Whittaker, 1982).

All specimens immersed in Powerade stained red (despite rinsing with DDW), confirming the observations of Vasan (1998), who found Powerade™ was more viscous than Gatorade™, Sports Plus™ and Coca Cola™. The more tooth-adhesive the drink, the greater the erosive potential (Milosevic, 1997).

Although adding 0.09-0.25% CPP-ACP to Powerade™ eliminated erosive steps, surface changes occurred. *In vitro*, dissolution of enamel prisms occurs sequentially as the erosivity increases, developing first in the prism sheaths, then in prism cores, and finally interprismatically (Meurman and ten Cate 1996; Grando et al., 1996). Following sheath dissolution, the heads and tails of enamel prisms dissolve and finally all prism structure disappears. In the present study, specimens in Powerade™ showed a consistent etch pattern with peripheral dissolution while prism cores remained intact. Specimens in P plus 0.063-0.25% CPP-ACP showed inconsistent etch patterns; areas of intact enamel were interspersed with superficial irregularities or globular deposits which increased with increasing CPP-ACP concentrations. These may represent enamel crystals, repair crystals or surface deposits subsequent to demineralisation or remineralisation processes, or precipitates from the high concentrations of calcium and phosphate held in surface proximity by CPP-ACP. Superficial calcium fluoride globules have been noted to a depth of about 40 µm in enamel following *in vitro* application of fluoride solutions (Øgaard, 2001; Petzold, 2001). The surface irregularities could also represent remineralisation of the smear layer created during polishing. Further studies are required to elucidate the nature of the surface and subsurface enamel.

Enamel polishing before profilometry allows measurement of tooth structure loss produced by limited amounts of acid (Hughes et al., 2000). The critical pH for dissolution of hydroxyapatite is 5.2-5.5 and below 4.5 for fluorapatite (Meurman and ten Cate, 1996). Polishing may remove surface fluorapatite and increase the critical pH for enamel dissolution, making it more erosion-susceptible than an intact surface (Zero, 1996; Hughes et al., 2000; Hunter et al., 2000). Polishing removes large crystallites and enamel high in carbonate and fluoride concentrations, exposing more uniform hydroxyapatite and allowing the formation of more uniform erosive lesions (Hunter et al., 2000). Thus *in vitro* erosion may exceed that expected *in vivo*.

With increasing CPP-ACP concentrations, the pH of Powerade™ increased and the titratable acidity decreased. Although pH values were below those critical for dissolution of hydroxyapatite and fluorapatite, it is speculated that erosion was limited by the high calcium and phosphate concentrations. Titratable acidity is a better indicator of erosivity than pH, representing the concentration of hydrogen ions that can dissociate in solution (Grenby et al., 1989; Milosevic, 1997). The lower titratable acidity and higher pH associated with adding CPP-ACP may have limited erosion. Further, CPP-ACP acts as a reservoir for calcium and phosphate ions; by maintaining these ions in a state of supersaturation with respect to enamel CPP-ACP decreases demineralisation and increases remineralization (Reynolds, 1998). As pH falls, CPP-ACP dissociates to form calcium and phosphate ions, minimising the pH drop and limiting demineralisation.

Modifying acidic drinks by supplementing with calcium, phosphate or milk products can reduce erosivity (Grenby, 1996; Hughes et al., 1999b; Larsen and Nyvad, 1999; Hughes et al., 2000). The reaction follows the Law of Mass Action, which states that "the rate of a chemical reaction (eg. dissolution of enamel mineral) is proportional to the concentration of the reacting substances (eg. calcium and/or phosphate) present at any given time" (Grenby, 1996). Therefore the presence of the reaction products of enamel dissolution decreases the rate and progression of enamel demineralisation.

Product modification should not alter product taste (Grenby, 1996; Hughes et al., 2000). The duo-trio taste test used in the present study is appropriate for small samples of untrained subjects (Meilgaard et al., 1991). Ten tasters could not differentiate Powerade™ from Powerade™ plus 0.125% CPP-ACP, and ten tasters correctly identified a difference but commented that the samples tasted very similar. Since a difference of 15 responses is required for statistical significance, it was concluded that the drinks did not differ in taste. The test is one of overall difference, chosen over taste acceptability tests as it is readily conducted, needs few subjects and requires no training. Other taste tests require larger sample sizes or formal training (Meilgaard et al., 1991). A trained panel should evaluate further the palatability of this modified product.

This *in vitro* study confirmed that Powerade™ erodes human enamel. Adding CPP-ACP (0.09% – 0.25%) eliminated the erosive step, but resulted in microscopic surface irregularities.

With increasing concentrations of CPP-ACP, the pH increased and titratable acidity decreased and erosivity decreased. The colour, clarity and taste of Powerade™ were unaffected by adding 0.125% CPP-ACP.

Acknowledgments

This research was supported by the Research Committee, School of Dental Science, University of Melbourne, and NHMRC Grant.

References

Coombes JS, Hamilton KL (2000). *The effectiveness of commercially available sports drinks.* Sports Med 29:181-209.

Grando LJ, Tames DR, Cardoso AC, Gabilan NH (1996). *In vitro study of enamel erosion caused by soft drinks and lemon juice in deciduous teeth analysed by stereomicroscopy and scanning electron microscopy.* Caries Res 30:373-378.

Grenby TH, Philips A, Desai T, Mistry M (1989). *Laboratory studies of the dental properties of soft drinks.* Br J Nutr 62:451-464.

Grenby TH (1996). *Lessening dental erosive potential by product modification.* Eur J Oral Sci 104: 221-228.

Hall AF, Buchanan CA, Millett DT, Creanor SL, Strang R, Foye RH (1999). *The effect of saliva on enamel and dentine erosion.* J of Dent 27:333-339.

Hughes JA, West NX, Parker DM, Newcombe RG, Addy M (1999a). *Development and evaluation of a low erosive blackcurrent juice in vitro and in situ. 1.* Comparison with orange juice. J of Dent 27:285-289.

Hughes JA, West NX, Parker DM, Newcombe RG, Addy M (1999b). *Development and evaluation of a low erosive blackcurrent juice drink 3. Final drink and concentrate, formulae comparisons in situ and overview of the concept.* J of Dent 27:345-350.

Hughes JA, West NX, Parker DM, van den Braak MH, Addy M (2000). *Effects of pH and concentration of citric, malic and lactic acids on enamel,* in vitro. J of Dent 28:147-152.

Hunter ML, West NX, Hughes JA, Newcombe RG, Addy M (2000). *Erosion of deciduous and permanent dental hard tissue in the oral environment.* J of Dent 28:257-263.

Imfeld T (1996). *Dental erosion. Definition, classification and links.* Eur J Oral Sci 104:151-155.

Jarvinen VK, Rytomaa II, Heinonen OP (1991). *Risk factors in dental erosion.* J Dent Res 70:942-947.

Larsen MJ, Nyvad B (1999). *Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate.* Caries Res 33:81-87.

Lussi A, Jaeggi T, Jaeggi-Scharer S (1995). *Prediction of the erosive potential of some beverages.* Caries Res 29:349-354.

Mathew T, Casamassimo PS, Hayes JR (2002). *Relationship between sports drinks and dental erosion in 304 university athletes in Columbus, Ohio, USA.* Caries Res 36:281-287.

Meilgaard M, Civille GV, Carr TB (1991). *Sensory evaluation techniques.* 2nd ed, Florida: CRC press, pp. 71-74, 339.

Meurman JH, Frank RM (1991). *Progression and surface ultrastructure of in vitro caused erosive lesions in human and bovine enamel.* Caries Res 25:1-6.

Meurman JH, ten Cate JM (1996). *Pathogenesis and modifying factors of dental erosion.* Eur J Oral Sci 104:199-206.

Milosevic A (1997). *Sports drinks hazard to teeth.* Br J Sports Med 31:28-30.

Nunn JH (1996). *Prevalence of dental erosion and the implications for oral health.* Eur J Oral Sci 104:156-161.

O'Dea J, Rawstone P (2000). *Consumption of dietary supplements and energy drinks by schoolchildren.* Med J Aust 173:389.

Øgaard B (2001). CaF2 formation: *Cariostatic properties and factors of enhancing the effect.* Caries Res 35 (suppl 1):40-44.

Petzold M (2001). *The influence of different fluoride compounds and treatment conditions on dental enamel: A descriptive in vitro study of the CaF2 precipitation and microstructure.* Caries Res 35 (suppl 1):45-51.

Reynolds EC (1998). *Anticariogenic complexes of amorphous calcium phosphate stabilised by casein phosphopeptides: A review.* J Spec Care Dent 18:8-16.

Silverstone LM, Saxton CA, Dogon IL, Fejerskov O (1975). *Variation in the pattern of acid etching of human dental enamel examined by scanning electron microscopy.* Caries Res 9:373-387.

Sirimaharaj V, Messer LB. Morgan M (2002). *Acidic diet and dental erosion among athletes.* Aust Dent J 47:228-236.

ten Cate JM, Imfeld T (1996). *Dental erosion, summary.* Eur J Oral Sci 104:241-244.

Vasan N (1998). *An in vitro investigation into the erosive potential of three popular sports drinks on human dental enamel* (MDSc thesis). Melbourne, Australia: University of Melbourne.

Whittaker DK (1982). *Structural variations in the surface zone of human tooth enamel observed by scanning electron microscopy.* Archs Oral Biol 27:383-92.

Zero DT (1996). *Aetiology of dental erosion-extrinsic factors.* Eur J Oral Sci 104:162-177.

Corresponding author

Professor Louise B Messer, School of Dental Science, The University of Melbourne

Email ljbm@unimelb.edu.au
Phone 03 9341 0292
Fax 03 9348 2415

Colgate® Corner

by Dr Jackie Robinson, Colgate Professional Relations Manager

Since the June edition of Synopsis, there has been lots of activity from Colgate!

New Colgate Oral Care brochures

In August, for Dental Awareness Month, Colgate launched three new Oral Care brochures:

- Infants and Toddlers
- Children 3 – 12
- Teens and 20's

Brochures may be requested through the Colgate Call Centre 1800 262 111.

FDI Congress 2003

We hope that those of you who attended the FDI Congress in Sydney enjoyed the event as much as we did at Colgate. Colgate had a significant presence at the Congress. On the Colgate stand, we gave out 4000 sample bags and recorded more than 22,000 "hits" on the touchscreens.

Over 1500 delegates and over 4500 children and parents visited the Colgate Bright Smiles, Bright Futures Activity Centre. The BSBF Centre was the only public event at FDI and drew large crowds to the weekend outdoor entertainment. The Colgate Whitening Symposium attracted over 650 delegates.

Nemo 3D brushes

I hope by now that all of you have taken the opportunity to see the fabulous Disney movie "Finding Nemo". The movie is such a joy and features not only Australian settings – the Great Barrier Reef and a dentist's office in Sydney – but also lots of Australian talent. To help your young patients join in the fun of Finding Nemo, Colgate is offering Nemo 3D toothbrushes. These brushes are available exclusively through dental clinics and practices and can be ordered through Halas Dental, telephone 1300 658 822 or fax 1300 658 810. Place your Christmas order now!

The changing patterns of chronic illness in children and the impact of this on their dental management

Winning 2002 Postgraduate essay
Anjella Stritharan

Introduction

Chronic illness refers to the irreversible presence, accumulation, or latency of disease states or impairments that involve the total human environment for supportive care, maintenance of function, and prevention of further disability.[1] It has been cited that some 10-20% of children will experience a chronic medical condition at some point in childhood, with 5-10% having a moderate to severely handicapping long-term illness or disability.[2] The number of people affected by chronic illness has been difficult to assess, because prevalence studies in the past have produced highly variable results due to problems of both definition and measurement. Chronic childhood illnesses that have received attention in the literature include: diabetes mellitus, nephrotic disorders, cystic fibrosis, arthritis, spina bifida, asthma and malignancies – to name a few. It is currently thought that chronic diseases of known or possible environmental origin are replacing infectious diseases of the past, as major the contributors to childhood illness.[3] Chronic illnesses in children also impact upon dental management at a number of levels. This paper will attempt to explore: the changing patterns of chronic illness in children – through the social context of illness, changing philosophies towards health care, and the role of the family/ care giver; the impact on dental management – with an emphasis on integrated treatment planning from both a medical and dental perspective, as well as issues related to the provision of health care; and finally directions for the future – with a particular focus on strategy planning and service provision.

Changing patterns of chronic illness in children

In the last few decades, care for people with chronic illness has risen high on the political, social and medical agenda. This has reflected a variety of trends such as demographic patterns, provision and use of public services, cultural assumptions about care, and the prevalence of chronic illness.[4] Chronic illnesses, may be thought of as those conditions that exhibit some long-term influence upon the lives of sufferers. They are generally conditions for which treatment of the underlying disease process is not available; the emphasis of care and rehabilitation is therefore more on enhancing and sustaining the quality and fullness of life, than on reordering the disease process.[4] The increasing strain for families, the health care system, social services, and the economy is likely to continue – even if the proportion of people with chronic illness is assumed to remain constant, because absolute numbers in the population will increase as a consequence of an aging population.[5] Advances in medical knowledge and technology will also mean that more people, who, in the past, would have been treated in a hospital setting will now present more frequently in general practice.[4] There is also likely to be an increased emphasis on palliative measures designed to improve quality of life – the implications of which are yet to be seen.[6] Therefore it is clear that the social context of chronic illness in children has undergone significant change, and will continue to do so.

A more holistic approach towards health care and patient management has become more widely accepted as well. Evaluating factors such as: the extent of disease and its complications in the child; the physical effects of the illness on the child; how the illness has affected the child's performance at home, at school and with peers; how the child has adjusted to the illness; the impact of the child's illness on the family and its members and how the family has adjusted to the special impact or burden of the illness – are now considered to be important as well.[2] Current opinion suggests that more effective management of a child with chronic illness is likely to occur when initial care and overall multidisciplinary treatment planning is coordinated by the paediatric doctor[7] – as attempts are made to confine the consequences of the condition to the minimum manifestation, encourage normal growth and development, assist the child in maximising his or her potential in all possible areas, and to prevent or diminish the behavioural and social consequences of a chronic condition.[2] The goal of having the affected individual occupying as normal a place in the life of the community has implications for many aspects of the patients life – from management of the treatment regimen and control of symptoms – to management of social isolation, changed vocational status and family relationships. This is associated with a reorientation of the focus of care from repairing damage caused by disease, to education and understanding for living with chronic illness. As a result of living in an age of changing consumer demands and expectations, there is also a need for providers to offer choices and link the experience of chronic illness with environmental conditions, material resources and the demands of contemporary culture and social structure.[4] Therefore a wide range of health-related disciplines must join forces if opportunities to reduce morbidity and mortality associated with chronic disease are to be realised.

It has also been increasingly recognised that family and social factors influence response to illness and subsequent outcome[8]; and that the family is a key resource in care and rehabilitation.[9,10] The family, has a central and enduring influence on children. Regardless of their composition, they are all subject to a variety of social forces that influence how well they are able to meet children's needs.[11] It is important to note that the experience of chronic illness may also lead to disadvantages not only for the patient, but for those close to them as well. The contribution of family and other informal carers has become the central guiding principle needed for successful community care in the development of health and social services for people with chronic illness.[4] However, social

trends indicate some uncertainty about the likelihood of increasing levels of previously assumed informal care. Specific areas that have been noted in the literature include – the decreasing level of fertility (fewer children), increasing geographical mobility of the population, growing emancipation of women (lessening their willingness and ability to provide informal aid) and moves towards a more individualistic lifestyle, decreasing the potential for family care – hence a move away from significant support from the traditional concepts of 'family' care.[5]

It is important to keep in mind that trends in 'family' care will depend greatly on changes in attitudes to care and to future values. In any case, understanding the functions families serve and the factors that constrain them, help in being able to assist parents in promoting their children's health and well being.[11] A sound, effective and ethical approach to chronic illness must lie in the awareness of and attention to the experiences, values, priorities and expectations of patients and their families. Therefore the future for community care and the implications for policy are clearly dramatic – as little is known about the attitudes to, or changing expectations for caring, and some of the problems for families that are associated with different chronic conditions.

Impact on dental management

Dental problems are amongst the more common health problems affecting children. As far as the practice of paediatric dentistry is concerned, it should be governed by a simple but fundamental philosophy – treat the patient not the tooth.[12] This is particularly relevant in the case of children with chronic illnesses. Implicit in this philosophy is a commitment to consider the child's feelings, to gain the child's confidence and cooperation, and to perform treatment in a kind, sympathetic manner, so as not to be concerned with only providing the treatment currently required, but also with promoting the child's future dental health by stimulating positive attitudes and behaviour regarding dental care.[12] It is critical that the dentist remains knowledgeable about the patient's medical condition/s because many disorders necessitate alterations in the provision of dental treatment, particularly in the case of chronic illness.[13] In addition to this many medical disorders have significant dental manifestations as part of the disease entity, or the required treatment regimen to control the physical symptoms of the disease. If risks to patients are to be avoided, essential areas of knowledge which the dentist must have include: current information on the medical status on the patient, understanding of the potential effects of interactions between the underlying medical condition and dental treatment and avoiding such interactions or to manage any interactions which may follow dental treatment.[13] Therefore it is mandatory that practitioners are aware of the current health status of all their patients so that this information is appropriately integrated into the patients treatment plan.

Primary care physicians providing health care for children can also prevent morbidity resulting from dental problems.[15] Common dental problems seen in children include dental caries, periodontal disease, malocclusion and trauma. Because there is no associated mortality or obvious serious physical morbidity, physicians may overlook the importance of dental problems in children. The medical practitioner can play an important role in the oral health of children with chronic illnesses from two perspectives.[16] Firstly, children usually see the paediatric doctor before they see the dentist, hence they play an especially important role in preventive oral health care in early childhood.

The appropriateness and effectiveness of preventive measures varies throughout the life of a child, and recommendations should be tailored to the needs of the individual and can contribute to the prevention of early dental caries through counselling practices. Second, as a respected and influential resource to parents, the paediatrician can also set the tone for the importance of oral health as a part of general health and lifestyle priorities.[16] Another important consideration is that oral pathology can have a significant effect on the progress of many systemic disorders and may influence the success or failure of the medical therapeutic program.[17] Oral health status can affect nutritional requirements for growth and development, progress of treatment, and even postsurgical healing.

It can contribute to the success of many surgically corrected congenital malformations and organ transplantations. Otherwise, poor oral health and neglect may adversely affect the outcome of surgical interventions and the progress of the disorder.[17] Therefore the primary care physician providing health care for children can help prevent the physical, psychosocial and economic consequences of dental problems by appropriate assessment, education and referral of children to the dentist.[15]

The actual provision of health care and practice environment, is another important area that warrants attention as well. The establishment of systems of services that reflect the principles of comprehensive, community based, coordinated, family centred care are essential for effectively fostering and facilitating activities to: avoid the initial occurrence of chronic and disabling conditions among children; reverse or slow the progress of such conditions; and minimise the complications and impact.[18] The establishment of these service systems is also essential to strengthen the ability of families to care and cope with such children and enables children with more serious conditions to be placed in home and community-based living arrangements rather than in institutionalised living arrangements.

As a consequence, this has a number of other implications. Because the majority of medically compromised patients need or want oral health care, a working knowledge of the multitude of compromised states is essential for dental professionals.[14] Their roles encompass recognising and understanding conditions that reflect compromised states, the presentation of adverse side effects of procedures and drugs used in dentistry, formulating treatment plans that are consistent with a patient's medical status, deciding whether referral is required, as well as the appropriate setting in which to carry out any treatment. Given the current practice environment for paediatric dentistry, there are likely to be issues with suitability of staffing in various dental practice settings, finance and insurance as well as medicolegal responsibilities, in the future.

Future directions

International research has shown that health systems can be designed to prevent and manage chronic diseases more effectively – however it is essential that system level of change is accompanied by, and supportive of the empowerment and active participation of individuals, their families and the community.[18] In the past there has been great dissatisfaction with disability services, which could reflect a poor system for the identification and management of problems of chronic illness in general practice.[19,20] Reports of unclear responsibilities, inadequate co-ordination and poor communication between general practice, hospital and community services, were common.[20,21] Formal services for care and rehabilitation may have been fragmented but nevertheless undermined the efforts of patients and their families, or was misdirected.[22] It is therefore important to appreciate the current situation as a basis for developing ideas, policies and services that are relevant and sensitive to the changing needs of people with chronic illness and their families.

Strategy planning involves ensuring an effective information base to guide action. As well as seeking to reduce people's exposure to risks, strategies aim to help individuals to develop personal skills to exercise more control over their own health and environments and to make better choices.[18]

Given the great range of influences on health, many major improvements require a strong partnership between public health and clinical care, and also that the health sector work with other sectors to make the best of limited resources. For example, the systematic surveillance of risk factors and their determinants, systematic development of the evidence base to inform policy and program design, evaluation and performance management; and strengthening prevention and health promotion, are important.[18] Other issues such as: reducing risk factors and their determinants; enhancing protective factors; promoting health across the life course; building partnerships for intersectoral action and supportive public policies; giving priority to populations most at risk; and improving systems of care for those with chronic disease – could help strengthen the role of prevention in the health care system – as well as improve early detection and intervention, integrated primary health care systems, partnerships and consumer participation.[18]

A strategic approach aims to build on current developments, recognising that a broader, systematic and collaborative prevention effort has the potential to significantly increase the impact on health outcomes. Health interventions can range from clinical and preventive attention to individuals, through to efforts to improve the physical, social and economic environment for special groups or the community as a whole. Opportunities are present in established settings for primary prevention, such as schools and the work place; in community-based services that can incorporate early intervention strategies; and in specialist and community care services where prevention efforts focus on disease management and continuing care.[18]

Education and advice through a supportive strategy also demands an understanding of the ideas, concerns and expectations of patients. Some indication of the character and proportion of these values and beliefs can be generated from research, which can offer a guide for public policy.[18] It is important to also keep in mind that improvements in health technology as well as an aging and more health conscious population – will lead to growing demands on health and treatment services.[23] Therefore planners and service providers in the future will still need to continue to identify lack of information about expectations, priorities and assessments held by patients and their families; and about the problems associated with everyday life with long-term illness.[24]

Conclusion

The complexity of issues surrounding the changing patterns of chronic illness in children, is thus apparent. The various trends in society, changing philosophies towards health care and recognition of the importance of family – have contributed to this. These changes have in turn had an impact on dental management, from the perspective of preventive treatment planning, but also with regards to actual service provision.

It will be interesting to see developments in the future given the current situation – as we still have a long way to go as far as addressing the key issues and establishing an adequate base for care.

References

1. Royal Australian College of General Practitioners (1997) *Curriculum Statement on Chronic Illness*, Melbourne
2. Rudolf,M (1999) *Paediatrics and child health* Blackwell Science Oxford Chapter 10
3. Australian Institute for Health and Welfare 2000. *Australia's health 2000: the seventh biennial health report of the Australian Institute of Health and Welfare*. Canberra AIHW (Chapter 8)
4. Anderson R, Bury M (1988) *Living with chronic illness :the experience of patients and their families* Unwin Hyman, Boston
5. Steering Committee on Future Health Scenarios (1987) *Growing old in the future* Martinus Nijhoff, Dordrecht
6. Hall RK (1979) *Dental management of the chronically ill child* Australian Dental Journal Oct; 24 (5):334-341
7. McInerny T (1984) *The Role of the General Pediatrician in Coordinating the Care of Children with Chronic Illness* Pediatric Clinics of North America W.B. Saunders Company, Philadelphia 31 (1) 199-210
8. Kasl S (1983) *Social and psychological factors affecting the course of disease: an epidemiological perspective* in D. Mechanic (ed) Handbook of Health, Health Care and the Health Professions Free Press, New York p683-708
9. Smith RT (1979) *The rehabilitations of the disabled: the role of social networks in the recovery process* International Rehabilitation Medicine 1:63-72
10. Anderson R (1987) *The unremitting burden on carers* British Medical Journal 294:73-74
11. Schor EL (1995) *The Influence of Families on Child Health: Family Behaviours and Child Outcomes* Pediatric Clinics of North America W.B. Saunders Company, Philadelphia 42 (1) 89-102
12. Mc Donald RE, Avery DR (1994) *Dentistry for the Child and Adolescent* Mosby, St Louis Chapter 23
13 Goss AN (1984) T*he Dental Management of Medically Compromised Patients* International Dental Journal 34:227-231
14. Pless IB (1984) *Current Controversies and Technical Advances* Pediatric Clinics of North America W.B. Saunders Company, Philadelphia 31 (1) 259-274
15. Howard BJ (1995) *The Referral Role of Pediatricians* Pediatric Clinics of North America W.B. Saunders Company, Philadelphia 42 (1) 103-118
16. Schaefer TE, Adair SM (2000) *Prevention of Dental Disease: The Role of the Pediatrician* Pediatric Clinics of North America W.B Saunders Company, Philadelphia 27 (5) 1021-1042
17. Casamassimo PS (2000) *Relationships between oral and systemic health Pediatric Clinics of North America* W.B. Saunders Company, Philadelphia 27 (5) 1149-1158

18. National Public Health Partnership. *Preventing Chronic Illness – A Strategic Framework – Background Paper*, October, 2001
19. Tulloch A (1985) *Prevalence of disability observed in an Oxfordshire practice* Journal of the Royal College of General Practitioners 35 :386-370
20. Royal College of Physicians (1986) *Physical Disability in 1986 and Beyond* Royal College of Physicians, London
21. Blaxter M (1976) *The Meaning of Disability* Heinemann, London
22. Elian M, Dean G (1983) *The use of health services by patients with multiple sclerosis*'Lancet (14 May) 1091-1093
23. Stein REK, Jessop DJ (1984) *General Issues in the Care of Children With Chronic Physical Conditions* Pediatric Clinics of North America W.B. Saunders Company, Philadelphia 31 (1) 189-198
24. Cohen WI (1995) *Family-Oriented Pediatric Care: Taking the Next Step* Pediatric Clinics of North America W.B. Saunders Company, Philadelphia 42 (1) 11-20

Continued from page 11...

- Not all provincial branches offer the categories which include individual membership of the International Association of Paediatric Dentistry. With these categories, provincial branches forward the names and subscriptions for such members to me, and then I communicate directly with the IAPD Secretariat to enrol such members.

- Not all provincial branches offer the associate membership categories.

- Each calendar year, it is recommended that provincial branches send out their membership renewal forms towards the end of the year (i.e. October or November). By doing this, it is hoped that memberships will all be renewed by the end of that year, or early in the following year. Provincial branch secretaries and/or treasurers can then have their membership lists and federal and IAPD subscriptions (if applicable) returned to me as early as possible in the new calendar year. This return early in the year is especially important when members are to be signed up with IAPD.

- A standard ANZSPD membership form has been developed. A copy of this can be obtained by contacting the ANZSPD Federal Vice President, Dr John Winters, by email at jwinters@swiftdsl.com.au John is also keeper of the mailing list for the Federal society.

- At the General Meeting of the Society in Brisbane in November 2002, the annual full and associate federal membership subscription was set at AU$40.00, with an additional AU$100.00 for those taking IAPD membership.

- IAPD membership entitles a member to an annual subscription to the International Journal of Paediatric Dentistry (six editions a year) plus a discounted registration cost to attend IAPD Congresses.

- The federal **mailing list** (as opposed to membership list) does contain the names of people (for instance from overseas) who are not ANZSPD members. Similarly, various institutions (for instance university dental libraries) are included in the mailing list.

Alistair Devlin

Coming events

- June 2004 – VII European Congress of the European Academy of Paediatric Dentistry, Fira Hotel, Barcelona

  The official language will be English. During the symposia, simultaneous translation to Spanish will be provided.

  www.eapd.gr for registration, abstract submission, and other information.

  Or contact:

  Cati Aurell
  McCann Meetings
  Josep Irla i Bosch, 5-7 Ent.
  Barcelona, Spain
  Phone: + 34 93 206 46 46
  Fax: + 34 93 204 97 32

AUSTRALIAN AND NEW ZEALAND SOCIETY OF PAEDIATRIC DENTISTRY

Federal President

Dr Christopher Olsen
Royal Dental Hospital of Melbourne
711 Elizabeth Street
MELBOURNE VIC 3000
Phone: 03 9341 0268

Federal Secretary Manager

Dr Alistair Devlin
57 Burroughs Road
KARRINYUP WA 6018

Branch Executives

Branch	President	Secretary	Federal Councillor
NSW	Dr Soni Stephen	Dr Erin Mahoney	Dr Kareen Mekertichian
Qld	Dr Laurie Bourke	Dr Matthew Fracaro	Dr Kerrod Hallett
SA	Dr Scott Smith	Dr Sue Springbett	Dr Vicki Farmer
Tas	Dr Tasha Dodd	Dr Wayne Ottaway	Dr Tasha Dodd
Vic	Dr Mala Desai	Dr Jodie Heap	Dr Chris Olsen
WA	Dr Tim Johnston	Dr Alistair Devlin	Dr John Winters
NZ	Dr Callum Durward	Dr Mary Anne Costelloe	Dr Callum Durward

Editor Synopses

Dr Karen Kan

Correspondence Synopses

Karen Kan
The Editor, Synopses
138 Harp Road
Kew VIC 3101
AUSTRALIA

Printing and Distribution

Colgate

Colgate Oral Care
Level 15, 345 George Street
Sydney NSW 2000
AUSTRALIA

Mailing List

The mailing list for the distribution of Synopses is maintained by Dr John Winters on behalf of the Federal Secretary/Manager of ANZSPD. It is compiled from information supplied by the Branch Secretaries. If there are errors in your mailing details, please contact Dr John Winters or your Branch Secretary. DO NOT contact Colgate for address correction.

Submissions

All text for inclusion in Synopses must be submitted to the editor on floppy disk, zip disk, CD, or by email. Both PC and Mac formats are accepted. Media will not be returned. Address email to kan@iprimus.com.au. Please enclose your contact details and email address with all submissions.

Deadline next issue

23 January 2004